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THE JOURNAL of the Oklahoma State Medical Association.

VOL. VI MUSKOGEE, OKLAHOMA, OCTOBER, 1913 No. 5

DR. CLAUDE A. THOMPSON, EDITOR-IN-CHIEF.

THE SURGICAL TREATMENT OF CANCER OF THE LOWER LIP, WITH A REPORT OF 199 CASES.*

E. H. Beckman, M. D., Surgeon to the Mayo Clinic, Rochester, Minnesota.

In considering the subject of cancer and its treatment, one must necessarily take into consideration certain facts. Cancer in the United States as well as in other civilized countries is apparently on the increase. If it is not actually on the increase, it is certainly true that an increased number of cases are reported to the various Boards of Health and an increased number of deaths are attributed to its cause.

In frequency tuberculosis and cancer are often compared. The anti-tuberculosis campaign is largely responsible for the present decrease in tuberculosis. A similar movement directed toward educating the public in the present knowledge of cancer would undoubtedly produce similar results.

We are still ignorant of the cause of cancer, but this does not prevent us from knowing a great deal concerning its manner of growth or how to cure it in the early stages. A cancer on the surface of the body apparently never develops except at a point subjected to continuous irritation for a considerable length of time. Cancers developing in cracks in the lip, about warts and moles or the edges of ulcers are familiar to every physician. There is much evidence to show that they develop in other parts of the body only at sites of prolonged irritation. As friction develops heat in machinery, so prolonged irritation develops cancer in the human body. Consequently every source of irritation should be eradicated from our bodies since they are fertile soil for the development of the disease. All cancers in the early stages are localized to small areas and while still localized can nearly always be cured.

**Read before the Oklahoma State Medical Association, Enid, May 13, 1913.*

Our present knowledge relative to the cure of cancer is about as follows: There is no medical cure for cancer and the doctor who treats a patient having cancer as a medical case and endeavors to cure him by drugs not only accepts a fee from his patient for which he gives nothing in return but in addition takes away that patient's best chance of recovery by allowing the disease to spread while he is watching for results.

Paste containing arsenic is one of the oldest methods of treatment for cancer. Arsenic has no selective action for cancer but has some affinity for pathologic tissues, largely because they contain more water and have less vitality than normal tissue. Its use is confined principally to cancer quacks who apply it for money instead of results and who cannot differentiate a lipoma from a malignant growth. People submit to its use only through ignorance.

The Roentgen ray has been tried in thousands of cases of cancer with doubtful results. In a cancer situated beneath the skin or on the mucous membrane its effect is worse than useless, since it does not cure and gives the patient and also the physician a false sense of security. The treatment does, however, often relieve the pain in rapidly growing cancer. It stimulates the formation of fibrous tissue and so delays dissemination. Small, superficial cancers of the skin can be cured in this manner, but there are other methods of treatment which are more efficient, more rapid, and do not carry with them the danger of an X-ray burn. Radium, which seemed to hold out so much hope as a cure, although having been tried for several years, is still of doubtful value. The one method of treatment which gives results sufficient to actually class it as a cure for cancer is surgery. It should be remembered, however, that it is not surgery alone but **early** surgery which gives the results. Other factors being equal, the number of surgical cures are in direct ratio to the stage of the disease.

It should be impressed upon the people that it is the disease and not the treatment which is dangerous. As has been stated so tersely by one of my colleagues: "The danger is not from operation but from delayed operation."

As I stated in a previous paper, the three important factors which contribute to the cure of cancer of the lip by present-day surgical measures are: (1) The opportunity for an early diagnosis, (2) the slight tendency for the growth to form metastasis in vital and deep-seated parts of the body, and (3) the accessibility for removal of the primary growth and the lymphatics first involved after the disease has progressed beyond the primary focus. Cancer of the lower lip is ideally situated for an early diagnosis. The patient is aware of a lump or an ulcer which does not heal in the very earliest stages of the disease. While one can be almost positively certain in regard to the diagnosis in most of these conditions, the only absolute proof is a microscopic examination by a competent pathologist. The day has passed when it is necessary to wait for glands to become involved or to try out the effect of potassium iodide on

these cases. Any physician in America, no matter how far from the regular centers of population, can take a specimen, send it to a pathologist and get a telegraphic report within a week. The important thing in these cases is time if we wish to cure our patient. The only cases that are hopeless are those who have delayed so long that an operation is no

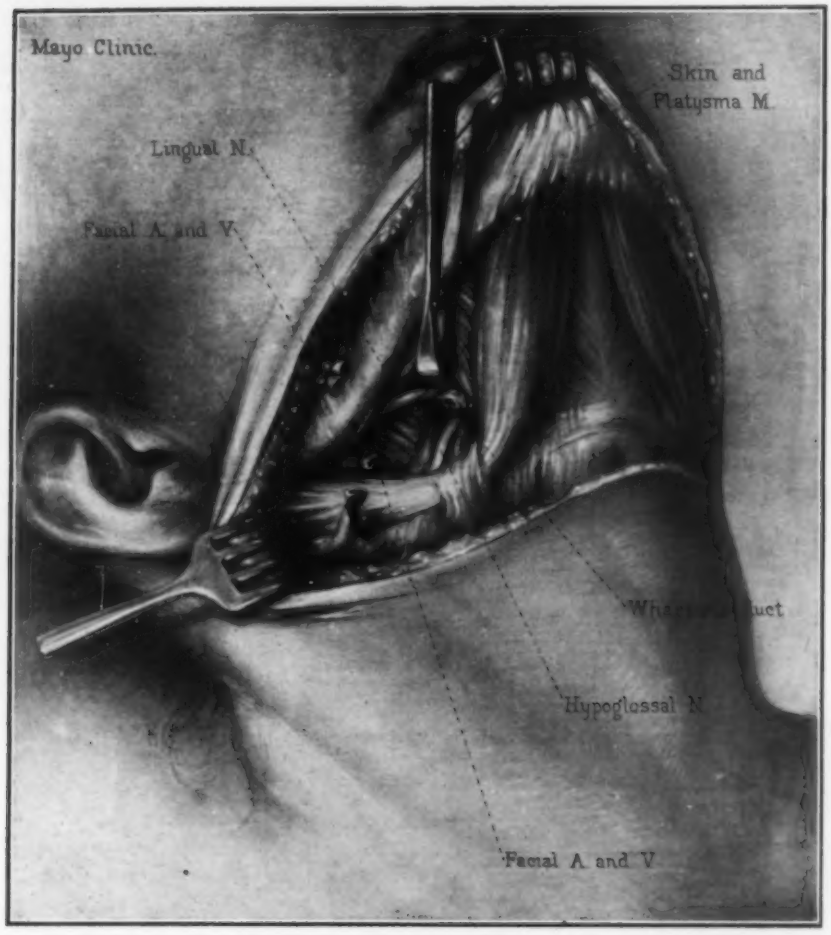

FIGURE I. Dissection of submental and submaxillary triangles for epthelioma of the lower lip.

longer advisable. A study of 4500 cases of cancer of the head and neck traced in the literature for Crile showed that in less than 1 per cent secondary cancer foci had been found in distant organs and tissues. That is to say, in cancer of the head and neck, death almost always occurs by local and regional development of the disease. The collar of lymphatics

in the neck forms a picket-line through which cancer is rarely allowed to pass. Every part of this lymphatic chain which surrounds the neck is easily accessible and can be removed with impunity. The medical profession have too long believed that cancer of the lower lip is not as malignant as cancer in other parts of the body. At the Mayo Clinic we have made it a practice for a good many years to do a radical operation on every case of any type occurring in this situation, no matter how early, provided the patient's condition would warrant such an operation.

The drainage area of the lower lip has been so carefully studied by Poirier, Cuneo, Kuttner and other observers that one knows definitely the lymph nodes which are first affected after the cancer has progressed beyond a local disease. The submental glands, lying in a triangle bounded by the anterior bellies of the digastric muscles and the hyoid bone drain the central portion of the lower lip. The submaxillary group, which drains the remainder of the lower lip as well as the cheek, lies in the submaxillary triangle. It sometimes happens that the submaxillary lymphatics on a side opposite the cancer will become involved. This rarely takes place, however, but one must remember that when the regular lymph channels are blocked, whether by cancer or inflammation, the lymph stream may flow in any direction similar to the blood, being taken up by the collateral circulation when the main trunk of a vessel is ligated. For these reasons it is advisable in every case to remove the lymphatics on each side at the primary operation. This should include the submaxillary salivary glands also, not because they become involved with cancer but because it is impossible thoroughly to remove the lymph nodes and leave the submaxillary salivary glands. After the glands from one side have been removed they should be examined immediately and, if found involved, the dissection should be carried down that side of the neck, so as to make a block dissection in which case the lymphatics removed must include both the anterior and posterior deep jugular group. If the glands on both sides in the submaxillary region be involved with cancer, nothing short of a block dissection on both sides of the neck is permissible.

The principle underlying the cure of cancer of the lower lip is the same as that involving the cure of cancer in other parts of the body, that is, the primary growth along with the glands into which the area of the growth drains must be thoroughly removed. There is one difference which we have observed in this connection. A cancer of the lower lip is either a local growth or else a metastasis in the adjacent glands. We have never observed a cancer occurring in the lymph vessels between the original growth and the neck; consequently, it does not seem necessary to remove the lymph vessels along with the glands and the primary growth. This is fortunate both for the patient and the surgeon, because it lessens the danger of infection from the mouth into the deep tissues of the neck. The original growth, however, should be removed with a wide margin, the wider the better.

In cases where only the submaxillary and submental regions have

been entered we do not believe that infection from the mouth is of serious consequence, since we have very often had to enter the mouth at the same

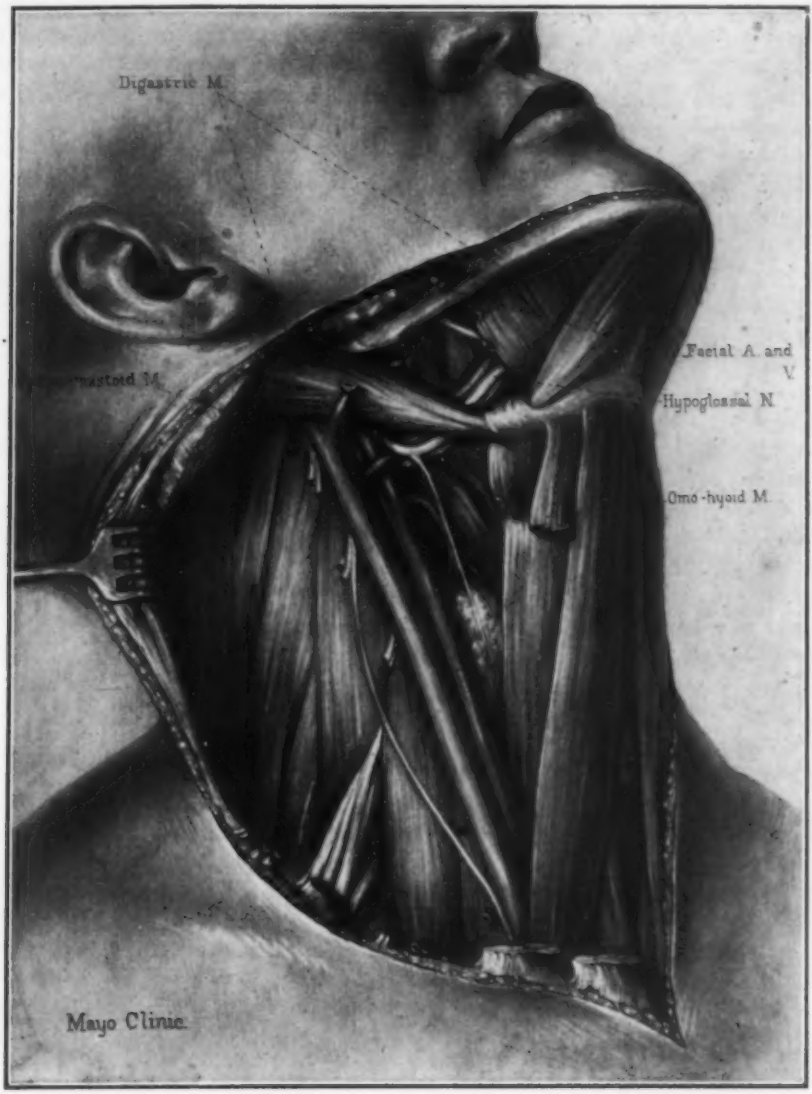

FIGURE II. Block dissection with removal of sterno-mastoid muscle, employed when submaxillary glands are carcinomatous.

time and also because infection often takes place through the cut duct of the submaxillary salivary gland. If it be necessary to extend the dissection down along the jugular to the clavicle, we prefer to perform the

operation in two stages, doing the neck part first, which blocks the spread of the disease, and operating on the primary growth one week later.

Our customary procedure in operating for cancer of the lower lip is illustrated by the accompanying charts. (Figs. 1-2-3). An incision is made ¾ inch below the ramus of the jaw from one sternomastoid muscle to the other. This incision extends through the skin and platysma muscle. The reason for making the incision as low as this is to avoid the small branch of the facial nerves which swings down below the angle of the jaw and then returns on the face to supply the muscles about the angle of the mouth. Where it is necessary to remove only a small portion of the middle of the lip a better cosmetic result is obtained by saving these branches of the facial nerves. If, however, it is necessary to remove more of the lower lip and widen the mouth by extending into the cheek it is not necessary to save these nerves. Through this incision the skin and platysma muscle are reflected down to the hyoid bone and up to the inferior maxilla.

All of the fascia and fat, including the submaxillary salivary glands, are removed from the submental and submaxillary triangles. It is necessary to ligate both the facial artery and vein. The blood supply to the face is so abundant through the other branches of the external carotid that we have never seen sloughing from this cause. The hypoglossal nerve and the lingual branch of the trifacial are exposed on each side and should be saved. After the removal of the glands this primary incision is closed, drainage being established through small separate incisions on each side. The platysma muscle is stitched first and then the skin with a subcutaneous suture. The wound in the neck is then carefully protected and the operation on the lip begun.

A quadrilateral section, including the full thickness of the entire lip, is removed, running down nearly to the point of the chin. The section removed should include the growth and from one-fourth to one-half inch on each side into the healthy tissues. The coronary artery should be tied on each side. An incision is then made from the lower angle of the quadrilateral parallel to the ramus of the jaw on either side as far as is necessary to obtain enough tissue to close the defect. The entire flap from which the new lower lip is to be made should be freed well from the bone. These flaps are then sutured together in the midline with silkworm gut sutures, the skin being approximated with horse-hair. If the skin remaining over the point of the chin is too long to be approximated to the new lip so that it produces wrinkles, it is shortened by taking out a small triangle at one or both ends of the incision, as shown by the accompanying diagram. This is the technic employed in cases where only a small portion of the lip is removed. If one-half or more of the lower lip must be removed, the primary incision is made in precisely the same way. In addition to the incision running from the lower end of the quadrilateral piece along the ramus of the jaw, it is now necessary to make incisions parallel to the former, extending from the corners of the mouth

directly into the cheek. These incisions should extend slightly downward rather than upward. The suggestion of J. Clark Stewart to incise through all the tissues except the mucous membrane, then go one-fourth inch higher before cutting through the mucous membrane is a valuable point in the technic, as it gives plenty of mucous membrane to stitch over the raw surface of the lower lip and thus prevents con-

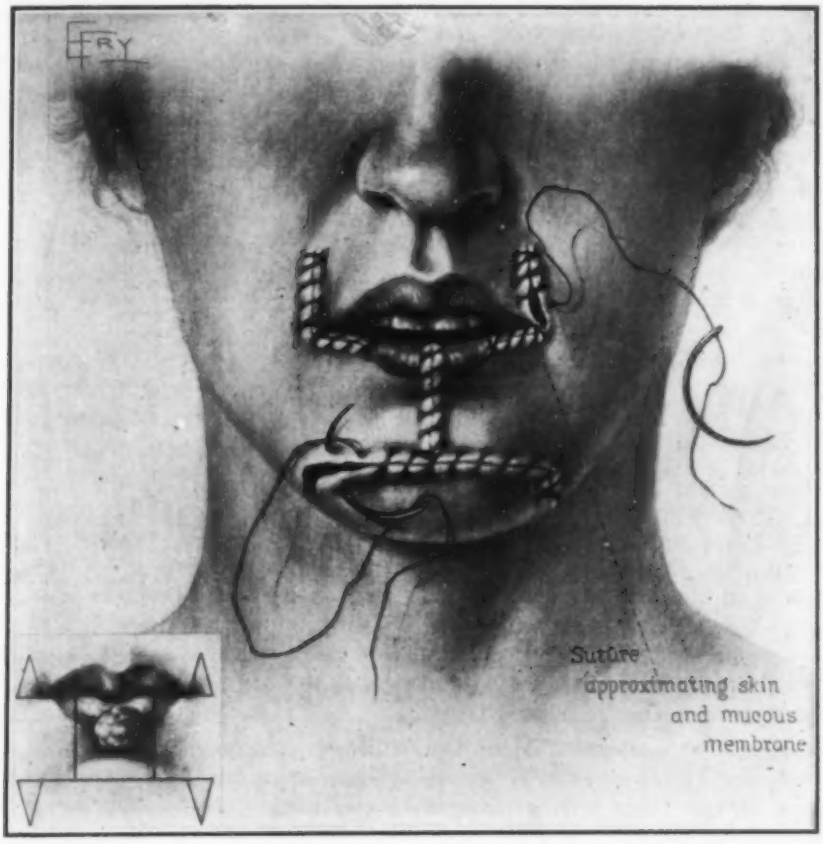

FIGURE III. Illustrating plastic operation for removal of a portion or the entire lower lip for carcinoma.

traction of the mouth later. When the flaps on each side are thoroughly free, they are approximated as in the former case, the only difference being that the raw surface of the lower lip must be covered with mucous membrane as just mentioned. It is now seen that the lower lip is shorter than the upper, and also shorter than the skin remaining on the chin. These two latter are shortened by removing triangular pieces from the extremity of each incision, as shown in the diagram. This plastic work

was taught me by C. H. Mayo and I have not seen it described in the literature.

From January 1, 1907, to December 31, 1911, there were 199 patients with cancer of the lower lip observed in the Mayo Clinic. This number includes only those patients having a cancer confined to the lower lip or one which had apparently originally arisen in that location. In addition to this number, there were a great many others having cancer of the upper lip, upper jaw, mouth, tongue, tonsils and pharnyx, which are not included in this report.

Twenty-five of these 199 cases were diagnosed from clinical findings only and consequently cannot be proved definitely to have been cancer. Two of the patients had the local growth removed from the lip before coming to our clinic. From the histories it appeared beyond question that the condition had been cancerous and it was thought advisable to remove the submental and submaxillary glands. This was done and both of the patients are well without any sign of recurrence. Seventeen patients in this series were considered inoperable when first seen. Two others refused operation and four are not accounted for, which means that we have no definite knowledge as to whether they refused operation or were considered inoperable. This makes up the group of 25 cases in which there was a clinical diagnosis alone. The remaining 174 cases have been proved to be cancer by both clinical and pathologic diagnosis. These 174 cases really form the basis of this report, since it is necessary to prove by microscopic examination that we are dealing with cancer.

One hundred twenty-six patients had a radical operation performed as the first operation. By a radical operation I mean that the local growth was excised with a wide margin and in addition the lymphatic glands which make up the drainage area of the lower lip were also removed either at the time of the operation on the lip or a few days later. Ninety-nine of these patients have been traced; we have received letters from them or examined them within the past few weeks. Twenty-seven of the group have not been heard from.

Of the 99 patients who have been heard from, 83 have no sign of a recurrence either locally or in the glands of the neck. Sixteen have either died of their original trouble or now have a recurrence. Seven of these 16 patients had glands involved at the time of their operation and three others had an extensive growth on the lip.

Considering only the cases of which we have a definite knowledge, that is, the 99 patients from whom we have received letters or who have been examined by us, 83 of which are cured, gives us a percentage of cures following a primary radical operation for cancer of the lower lip of 83.8 per cent. It might be interesting to note the time since the operation on these 83 cured cases: Two of them were just one year ago; 25, between one and two years; 17 between two and three years; 20, between three and four years; 15 between four and five years; and 4, over five years. In 18 of the above number glandular involvement was demon-

strated by the microscope at the time of their operation and 9 of these, or 50 per cent, are among the cured cases.

In another group I have placed 25 patients who had a late radical operation, that is, removal of the glands of the neck following one or two local operations on the lip or following treatment by paste. Twenty of these patients have been traced. Fourteen are classified as cured and six as not cured, giving a percentage of 70 per cent of cures in those patients having a late radical operation as compared with 83.6 per cent of cures in cases that had radical operations as the primary treatment.

Twelve of the above 25 patients had glandular involvement demonstrated by the microscope at the time of the operation; 4 of these are cured. While this group of 25 is much smaller than the previous group, it is interesting to note that when the radical operation was delayed only a third of the patients having glandular involvement were cured as compared to 50 per cent of cures in those having glandular involvement following a primary radical operation.

Five patients had an incomplete removal of the glands either as a primary or secondary operation. All of these patients have been traced. In this group are cases in which an operation was attempted but abandoned because the involvement was found to be so extensive. It also includes these cases in which glands were removed from but one side of the neck, the growth in the lip being confined entirely to one side. Two of these patients are well with no signs of recurrence.

In a last group consisting of 18 cases we have placed those patients who had an excision of the local growth without the removal of any glands. Most of these patients were seen early in the disease but their general condition or age prevented a radical operation. Fifteen of these cases have been traced, 11 of which are cured, giving a percentage of 73.3 cures. It has been stated by other observers or borne out by statistics that an early operation for cancer, although not a radical one, is often more favorable than a late radical operation. This holds true in the present series, as 73.3 per cent of the patients having an early local excision were cured as compared with 70 per cent of cures among those who had a radical operation in a late stage of the disease. There were no operative deaths in this series of cases.

Note.—I am indebted to my assistant, Dr. Ethan Flagg Butler, for compiling the statistics.

CANCER OF THE LOWER LIP.

GROUP	No. Cases	No. Operated	Traced	Not Traced	Cured	Not Cured	Inoperable	Per ct Cured
I. Clinical diagnosis only....	25	2	6	19	2	23	17	
II. Primary radical operation	126	126	99	27	83	16		83.8%
Glands involved	18	18	18		9	9		50. %
III. Late radical operation..	25	25	20	5	14	6		70. %
Glands involved	12	12	12	0	4	8		33⅓%
IV. Glands removed one side or incomplete	5	5	5	0	2	3		40. %
V. Local incision only.....	18	18	15	3	11	6		73.3%

ENDOCARDITIS.

P. P. Nesbitt, M. D., Muskogee, Oklahoma.

The term endocarditis includes all inflammatory affections of the endocardium. For convenience, these are subdivided into acute and chronic endocarditis. The acute is further divided into benign and malignant and these still further classified according to pathology, bacteriology and symptoms. Chronic endocarditis is practically synonymous with chronic valvular disease and as that is to be the subject of another paper to be read, will not be considered here.

The initial attack of endocarditis practically always develops as a complication of other disease. Later we may have attacks where it seems to be the only lesion, the disease which it originally complicated having entirely disappeared. The diseases most frequently complicated by endocarditis are (1) acute articular rheumatism, (2) chlorosis, (3) scarlet fever, (4) gonorrhea, particularly gonorrheal arthritis, (5) tonsilitis, (6) pneumonia, (7) puerperal sepsis, (8) infected wounds. Less frequently it complicates typhoid, diphtheria and other infectious diseases.

The classification of acute endocarditis as benign and malignant is purely arbitrary as they are the same process, differing only in intensity.

Pathology. The pathology of acute benign or simple or vegetative endocarditis is the development of vegetations on the endocardium and is almost entirely confined to the valves. These vegetations are due to bacteria and are verrucose, wart-like, papular, or pedunculated. At first they are grayish, gelatinous and transparent. They are composed of two layers, the deeper consisting of granulating endothelial and subendothelial tissue and the upper layer consisting of thrombi from the blood with which they come in contact. The thrombi contain few or no bacteria. The thrombi may become dislodged forming emboli in any part of the body. The effect of these is mechanical only, as they are not infective. Later the deeper layer becomes white and of fibrous consistency causing the valve to become sclerosed and often the orifice to become stenosed.

The pathology of malignant or ulcerative endocarditis is the development of an ulcerative process on the vegetations or the sclerosed valves of a simple endocarditis. The ulceration is usually confined to the valve but sometimes extends from the aortic valve to the aorta, from the pulmonary valve along the pulmonary artery as far as the hilum of the lung, from the mitral valve to the chorda-tendeneae or to the wall of the heart. In the aorta this may produce aneurysm, in the heart may destroy chorda-tendeneae, or may extend into the heart muscle deep enough to produce an opening between the ventricles or a rupture of the external walls of the heart. On the valves when the outer layers have been destroyed the pressure of the blood often causes stretching of the thinned walls producing valvular aneurysms. When the whole thickness of the valve is destroyed, of course, a leakage at once results usually sufficient to destroy compensation.

Thrombi form on the ulcerations containing a large number of bacteria. These are constantly being carried away in the blood stream causing a bacteremia. Pieces of the thrombi of variable size are often broken loose and are carried in the blood lodging in the different organs causing endarteritis, or embolism with infarcts. The results of these vary with the character of the material forming the embolism. If due to streptococci, a hemorrhagic infarct usually results; if due to staphylococci or pneumococci, a suppurative infarct develops. There is always present a polymorphonuclear leucocytosis and cultures of the infective organism may be made from the blood if a sufficient quantity is used.

Symptoms. The symptoms of simple endocarditis are a slight temperature rarely exceeding 101 F., heart rate nearly always affected, usually quickened but occasionally slowed, and often irregular. There is rarely any pain but there may be some palpitation and pre-cordial distress. These symptoms are not diagnostic of endocarditis and are more apt to be due to a myocarditis or pericarditis. The valve sounds, which must be depended on for making a diagnosis, are at first normal but as the disease progresses we get a muffling or irregularity of the cardiac sounds, later a murmur and still later the signs of valvular insufficiency. Diastolic murmurs are more suggestive than systolic for they are rarely functional. Embolism, when it occurs, is very distinctive.

The symptoms of malignant endocarditis are even more variable than those of the simple variety. Fever is a fairly constant symptom but it may be of almost any variety; constant, regular or irregular remittent, regular or irregular intermittent. In the so-called typhoid type, the fever may resemble very closely that of typhoid. In fact all the clinical symptoms of typhoid may be present including the typhoid tongue, rose-spots, relaxed bowels, hemorrhage from the bowels, and the diazo reaction may be present. The Widal reaction, of course, is absent and there is a moderate or severe polymorphonuclear leucocytosis and these alone enable us to differentiate a case showing these symptoms from a typhoid. Rigors followed by rapid rise of temperature, which continues high for awhile, then falls rapidly accompanied by free sweating is a very common type. These paroxysms are usually very irregular as to time of occurrence, but sometimes simulate quotidian or tertian malaria so closely that a blood examination is necessary to differentiate. Embolisms are very frequent and when they occur anywhere except in the lung are very suggestive of endocarditis of the left heart. When they occur in the lung they may be due to an endocarditis of the right heart or a dislodged thrombus from any part of the venous system. The symptoms and results of these emboli vary with the size and location and the infective organism present. If they occur in the brain we have a paralysis which may be more or less permanent or entirely disappear, or may result in brain abscess or suppurative meningitis. If they occur in the lung, spleen or liver there is sudden pain in the affected part, later the symptoms may disappear or there may be suppuration or gangrene in the part supplied by the plugged

artery. Emboli in the kidney are apt to cause blood or albumen in the urine and may result in suppuration or any form of nephritis. If they occur in the vessels of the skin they produce hemorrhagic or suppurative infarcts and by these we are often able to complete the diagnosis. On physical examination the area of cardiac dullness is increased, extending usually to the right of the sternum, the apex beat which at first is located normally travels outward and downward, often being well to the left of the nipple line and in the sixth or seventh interspace. The cardiac sounds change with the progress of the disease, the location of the changing sounds depending on which valve is affected. A post-systolic murmur heard best in the third interspace close to the sternum is indicative of affection of the aortic valve. If the mitral valve is involved the murmur is heard best at the apex. The first sound becomes prolonged and roughened, later a doubled sound appears, and still later the first sound is followed or replaced by a soft blowing murmur. Cabot states the murmurs most suggestive of endocarditis are those that change rapidly under observation, especially diastolic murmurs. There is usually some palpitation and pain or distress over the pre-cordium. General weakness and marked symptoms of sepsis are nearly always present.

Diagnosis. From the large number and variability of the symptoms enumerated it can be readily understood why there are so many mistakes and failures in diagnosing endocarditis. This was stated by Bamberger in the following words: "The diagnosis of acute endocarditis is seldom easy, usually difficult, and often impossible." The onset of the disease does not help much as it may be sudden or very insiduous. When in the course of an acute articular rheumatism, chorea, tonsilitis, scarlet fever, gonorrheal arthritis, or other acute infections, a disturbance of the heart action develops with an elevation of temperature without a corresponding aggravation of the other symptoms, we should suspect endocarditis and watch the heart very carefully. "When fever without known cause is present any cardiac murmur must be thought of in the light of a possible endocarditis." (Cabot.) When fever is accompanied by the characteristic changes in the size and location of the cardiac dullness and changes in the size and location of the cardiac dullness and changes in the heart the heart sounds and heart rate develop while under observation, we can be fairly sure that an acute endocarditis is present. The occurrence of emboli in different organs of the body is very distinctive and often enables us to make a positive diagnosis.

In differentiating between vegetative and ulcerative endocarditis the accompanying disease must be considered as well as the character and severity of the symptoms present. Ulcerative endocarditis is very rare in chlorosis, tuberculosis, typhoid, diphtheria and scarlet fever. Is rare in rheumatism and tonsilitis; frequent in the endocarditis of pneumonia, less frequent in those of erysipelas, septicemia, puerperal fever and gonorrhea. Sepsis is very prominent in the ulcerative form and much less

so in the vegetative type. Leucocytosis is more marked and bacteria are present in the blood in ulcerative endocarditis.

In differentiating the typhoid type of acute endocarditis from typhoid fever a blood examination is often necessary, as the clinical symptoms may be identical. In ulcerative endocarditis with remittent or intermittent temperature with rigors and sweats a blood examination may be necessary to differentiate from malaria, especially if the changes of temperature have any regularity. The blood here will show the absence of the plasmodia of malaria and the presence of leucocytosis. When the symptoms are not very marked they are often masked by the disease the endocarditis complicates and its existence is overlooked. The most frequent mistake is in diagnosing other forms of sepsis, myocarditis, and pericarditis as endocarditis. So that even where all aids to diagnosis are used and the utmost care taken autopsy shows many cases of acute endocarditis which were not suspected and many cases diagnosed as such to be some other affection.

Prognosis. The prognosis of simple vegetative endocarditis is good as to life but complete recovery is rare. When we consider that most cases result in chronic valvular heart trouble, often with stenosis, and consider the dangers from resulting incompetency and recurring attacks of endocarditis, simple or ulcerative, it is readily seen that the term "benign" endocarditis is really a misnomer. The prognosis of ulcerative endocarditis is always very grave. The result is usually death in from a few days to a few weeks but sometimes is prolonged to a year or more. The cases which recover are mostly from among those due to gonococcic infection.

Treatment. The treatment of acute endocarditis of all forms may be considered under two heads (1) prophylactic, (2) treatment of the disease after it has developed.

Prophylaxis resolves itself into (a) treatment of the causal disease, including the treatment of sepsis, (b) rest, (c) treatment directed to the heart when it shows irritability or other signs of becoming affected.

As our knowledge of the treatment of acute infections by serums and vaccines increases, the number of cases of endocarditis resulting from these affections will decrease. The casual diseases will be prevented, or their severity lessened, sepsis avoided or lessened, and the infected organism destroyed before such complications as acute endocarditis develop. At present, however, this is largely a dream and while some good results have been attained by these measures, our main reliance for prophylaxis is on absolute rest during the course of the infections that are liable to be complicated by endocarditis, and for several weeks after the temperature is normal in acute articular rheumatism and scarlet fever. Not only should the patient be kept in bed, but every effort should be made to keep the mind at rest also. The work of the digestive organs should be reduced by a light, easily-digested diet, largely liquid, care being taken to not give enough liquids to throw additional work on the heart and kidneys. Irritability of the heart may be quieted and slowed by use of the ice-bag.

Mustard over the heart, care being taken not to blister, often gives good results. Stimulants should be avoided, especially during the early stages of the infection, but later coffee, ammonia, whiskey or brandy may be given. Digitalis is not to be used unless tumultuous action of the heart threatens to produce embolism. Even then small doses of morphine may be more satisfactory.

In a person who has had an endocarditis resulting in injury to the valves, care should be taken to avoid exercises that will bring an unusual strain on the heart, and such a person should be kept in bed when suffering from any infection.

Treatment of an acute endocarditis after it has developed is specific or symptomatic. Specific treatment is still in the early stages but the serum and vaccine treatments, especially that of autogenous vaccine, has given some very promising results. Where the serum is used, it should be used in larger amounts and more frequently than in other affections. Symptomatic treatment must, of course, be governed by the symptoms present. Hypnotics and coal-tar antipyretics should be avoided. If a septic diarrhoea is present, it should not be checked at once.

After the acute stage of vegetative endocarditis is passed the iodides should be given to aid resolution of the vegetations.

THE DIAGNOSIS AND PROGNOSIS OF VALVULAR HEART LESIONS.

Dr. R. W. Williams, Anadarko, Okla.

In connection with valvular heart lesions we are always confronted with the question: "What is the seriousness of the trouble?" Naturally, this makes the prognosis of heart lesions one of the most interesting, and at the same time distressing, problems with which we have to contend. First, our patient will want to know: "Have I heart disease," as he calls it, "and if so, is it dangerous to life?" Entirely apart from this anxiety of our patient, and considered as of merely scientific interest, the prognosis should interest the physician as much as, or perhaps more, than any question that may arise, not excepting the treatment of the case.

It is utterly impossible and manifestly so to foretell the limit of life in any one case of heart disease. The utmost within our power is to group the cases roughly, and by considering carefully the various factors, arrive at an opinion that in the individual case, may come far short of the mark, but in the average, will be not far from right. It is our purpose, in this paper, therefore, to analyze these various factors. It would be preposterous for us to attempt a prognosis, without endeavoring to make an accurate diagnosis of our pathological condition. The diagnosis should establish four points, viz.:

First. The valve involved and the lesion present, i. e., an insufficiency or a stenosis.

Second. The extent of the lesion.

Third. Presence or absence of compensation.

Fourth. The Etiological factor.

The diagnosis and the character of the lesion is often a relatively simple matter, as this depends on well understood data. At times, however, when we have a combination of lesions or a very slight lesion, a diagnosis may be very difficult, and a variety of opinions will be expressed by as many men. In the combined lesions, the important elements are usually recognized; the chief difference of opinion being in regard to slight lesions, complicating more severe ones where the question arises as to whether or not a lesion exists. The ability to diagnose and diagnosis itself are very important.

Perhaps the most frequent question is in regard to mitral insufficiency, and right here, may we say emphatically, that a murmur in itself, is not sufficient evidence to make a diagnosis on. There must be some displacement of the apex outward or other evidence of enlargement of the heart. And, unless there be some accentuation of the pulmonary second sound, there is probably no lesion present in spite of a murmur. Anemia will often produce such a murmur. And these so-called haemic murmurs are not evidence of organic trouble. An anatomical peculiarity in the chordae tendinae may cause the first tone to be muffled. According to my observations, these murmurs are most often heard at the base of the heart, just to the left of the sternum in the third inter-space. They may be heard at the apex. In children, we must not forget that the second pulmonic is often louder than the aortic. Often times also, in children, when we notice this apparent accentuation associated with a systolic murmur, it is necessary to withhold our opinion till time shows us that the murmur is functional..

The second aortic tone is often accentuated in arterio-sclerosis, or any other condition producing high arterial tensions, and this may cause us to overlook a mitral leak. Such a murmur will often entirely disappear on rest in bed, but it may easily be brought back by exercise; not invariably, however.

A man has a systolic thrill and murmur over the aortic area, and we err in diagnosing an aortic stenosis. This latter lesion does occur, but remember it is a rare condition, and should not be diagnosed unless other signs are present, such as an absence of the second aortic and slowly rising and long-sustained pulse wave. The systolic thrill and murmur found in the aortic area are most often due to aortic insufficiency. It is the result, possibly, of arterio-sclerosis of the valve, or to an aneurysm; only rarely are they due to an aortic stenosis.

A case under my care at one time showed strong evidence of broken compensation; there was no murmur, or at most a slight systolic murmur, which I interpreted to be due to a relative insufficiency. But when my patient died, and I carefully reviewed his history and remembered his small, hard pulse, the high area of dullness due undoubtedly to the dilation of the left auricle, and a peculiar slapping quality of the first tone,

I got to thinking and soon realized my man had died with a high grade mitral stenosis. On the whole, I believe we may say these cases are relatively easy to diagnose, but there are opportunities for error.

The extent of the lesion—

Although it is often difficult to accurately determine the extent of a lesion, it is perhaps more important to do so, than it is to establish the mere diagnosis or presence of the lesion; murmurs offer us practically no aid in determining the extent of a lesion except that as a rule a loud murmur indicates a slight lesion, and a relatively good condition of the heart muscles. When this loud murmur becomes weaker, however, it indicates as a rule, muscular weakness. Therefore, we need to look to other sources for our chief evidence and right here may we emphasize the importance of the examination of the apex beat by palpation. It is true, very often we are unable to locate the apex beat definitely and must rely on percussion, but when the other is possible, and when we can define it accurately, we have definite and irreproachable evidence of the exact condition of the left ventricle, since it is possible to tell by the width of the apex whether or not this cavity is dilated and by the force of the impulse whether there is hypertrophy present and to a degree how much. The general precordial impulse and the epigastric pulsation give us information as to the right ventricle, which is, of course substantiated by our findings on percussion; we may diagnose right ventricle hypertrophy on an accentuation of the second pulmonic, for this indicates increased tension in the pulmonary circuit and that in turn means more work for the right ventricle, which must have hypertrophied in order to do its work. So, judging from the size of the heart and the inference derived therefrom, we may diagnose the extent of a lesion. If there happens to be an associated myocarditis, a considerable dilitation may accompany a slight lesion. In these cases we may make an error in diagnosis, but the prognosis will be the same. As a rule, a large heart means an extensive lesion, and a slight change in the size of the heart means a slight lesion. An additional question as to the stationary or progressive character of the lesion comes up. We may say as a rule an insufficiency is apt to be stationary, while stenosis is more likely to progress. Other factors enter here which will be discussed later.

Compensation. We must decide whether or not compensation is perfectly established in each case. It is possible usually to establish this point by carefully eliciting the patient's history. If at any time he has suffered with shortness of breath on exertion, together with cough, etc., then probably compensation has been imperfect or has been on the verge of breaking. We must consider the various lesions separately upon our examination. We know that with mitral insufficiency we have hypertrophy and dilitation of the left ventricle, dilitation of the left auricle, increased tension of the pulmonary circuit and a consequent hypertrophy of the right ventricle. This sequence of pathological conditions gradually develop. Dilitation of the right ventricle is not a necessary part of the

picture, but when it does occur it means a broken compensation. At the bedside, when we have such a lesion, we will find the right border displaced to the right, while simple hypertrophy will not accomplish this displacement to any extent. Therefore, this displacement to the right of the sternum justifies a conclusion that we have a broken, or at least an imperfect compensation. With a mitral stenosis, we always have some insufficiency, and a high-grade stenosis is never perfectly compensated. We have in both mitral lesions, practically the same clinical findings, i. e., pulmonary passive congestion, evidenced by a bronchitis, oedema, etc., passive congestion in the portal circuit evidenced by a large tender liver and ascites; and passive systemic venous congestion, evidenced by oedema of scrotum, legs, etc.

Aortic lesions, when compensation fails, show the same symptom and signs of mitral lesions. With dilitation of the left ventricle, a relative mitral leak may develop, or the symptoms of a broken compensation may develop without there being any relative valvular leak. When we have both aortic and mitral leaks present it is often difficult to say whether the mitral insufficiency is primary or is a relative lesion due to the dilitation. This is often impossible to determine since in cases due to arteriosclerosis of the aortic valve, the anterior cusp of the mitral valve is often affected. It is well to remember that an acute endocarditis almost never causes an aortic leak alone, but that the mitral valve is also involved in the majority of cases.

Etiological Factor. Our pathology teaches us that there are two important groups of cases of valvular disease. In the one, where an acute endocarditis has occurred, the valve leaflets are thickened and retracted from scar contraction, and the chordae tendinae are thickened and shortened. Frequently the cusps of the valve are adherent. These changes occur on the free edges of the valves, the attached margin remaining unchanged.

In the other group, the valve changes depend on the primary changes in the aorta, and occur by a process of extension from the aorta. Hence the attached margin is thickened and often indeed calcareous. The valve segments become thickened and do not coapt perfectly, or the opening may be stenosed. Naturally, the aortic valve is most frequently involved in this hardening process, though the mitral may also be affected, owing to the fact that the anterior cusp arises from the aortic ring. On the other hand, the mitral valve is most frequently involved in the acute endocarditis process. The cause of acute endocarditis is very frequently acute articular rheumatism, although of course, it may be the result of scarlet fever, measles, septicemia, pneumonia and other acute infectious diseases. True, too, it is only too often that we are unable to elicit any cause for the trouble, and yet we have every evidence of the true nature of the malady.

There are also many obscure infections with which we have to deal, the true nature of which we do not recognize until we notice a subsequent

development of a valvular lesion Many of these cases give a history of "malaria" or some other acute disease that was nothing more nor less than an acute endocarditis that was not recognized. As stated above, atheromatous degeneration of the aorta seems ofttimes to extend to the aortic valve, thus causing valvular lesions. Every etiological factor favorable to the development of arterio-sclerosis is, therefore, important in the production of valvular disease. These include syphilis, alcoholism, lead, advanced life, excessive physical labor, etc. There is today unimpeachable evidence of the influence of chronic nephritis upon the development of these lesions.

It is of utmost importance to recognize these two groups of cases, for the prognosis is much different. In the primary sclerotic group the vessels of the heart itself are often involved with the production of endocarditis, angina pectoris, etc., and the patient may drop dead at anytime from an acute dilitation of the heart. Also, the lesion is more apt to be progressive. These cases occur as a rule, later in life, from forty years up, but do not forget that they may occur earlier. A tendency to an early sclerosis frequently runs in families, and we get a history of father, grandfather, uncles, etc., dying at 40, 50, 60 odd years of apoplexy, "dropsy," angina pectoris or other manifestations of vascular mischief. In the group due to a primary acute endocarditis, the lesion is less apt to progress, the heart muscle is not so often involved and the condition occurs more often early in life; as we know, many of these cases do recover, or at least the pathological condition remains quiescent for a considerable length of time; however, some of them die while yet in their prime.

Having made a diagnosis, not only of the lesion but also of its extent, the degree of compensation and the cause of the lesion, the prognosis of the individual case requires our attention, and right here we meet up with a large variety of opinions. Mitral insufficiency undoubtedly offers the best prognosis, and all in all aortic insufficiency the poorest. However, it is possible to qualify the latter statement, because if we separate this group as to cause, we find that the cases due to a primary acute endocarditis offer almost as good a prognosis as does mitral insufficiency. Both aortic and mitral stenosis are more severe, and again we must separate the slight from the grave instances of mitral stenosis. The milder cases of the latter where there are no pulse changes, but the predominating condition is the insufficiency that is always present, offer nearly as good a prognosis as mitral insufficiency alone. The severe cases on the other hand, offer almost as serious a prognosis as does aortic insufficiency due to arterio sclerosis.

Let us arrange the lesions then in the following manner of increasing danger:

1. Mitral insufficiency.
2. Mitral stenosis of slight degree.
3. Aortic insufficiency due to acute endocarditis.

4. Aortic stenosis.
5. Mitral stenosis of a high grade.
6. Aortic insufficiency due to primary chronic endocarditis.

We have observed that many of the cases of aortic insufficiency show more or less mitral insufficiency, and that practically every case of mitral stenosis shows more or less insufficiency. In neither of these cases does the mitral leak affect the prognosis very much. There is frequently found a presystolic murmur just external to the apex in cases of combined aortic and mitral insufficiency, and we wonder whether this murmur indicates a mitral stenosis. Frequently the only decision that can be arrived at is that if present, it is very slight. Where tricuspid insufficiency is diagnosed together with a mitral, or aortic and mitral lesion, it would be hard to say whether this is a primary or a relative lesion, but with improvement, if the tricuspid insufficiency disappears, we may diagnose a relative lesion positively, and we must remember that this is a very rare primary lesion and should almost never be diagnosed, particularly when compensation is imperfect. However, when partial stasis is much more extensive than is general systemic stasis, then we should suspect a primary tricuspid lesion. This marked disproportion occurs chiefly in right lesions, whether due to valve disturbance or to passive lung congestion due to "emphysema." It also occurs in adherent pericardium.

The extent of the lesion affects the prognosis directly, i. e., the more extensive the lesion, the poorer the prognosis.

Uncompensated lesions offer a poorer prognosis, of course, than do compensated lesions. We cannot always say that a broken compensation savors of death. If it occurs after very severe exertion, as dancing, the heart may once more regain its tone; while if the break occurs in the course of ordinary pursuits, the prognosis is of course grave. However, we sometimes have contradictory evidence to this rule. I well remember a case, following acute rheumatism, which we had watched develop mitral and aortic insufficiency. The patient was kept very quiet until his symptoms improved. One day he walked out for a little exercise. He suddenly became faint and showed a rapid, irregular pulse, with some evidence of dilitation. He soon improved, however, and insisted on returning to his home, some miles away. He was instructed to remain quiet for several weeks before resuming his work, that of a coal miner. We saw him three months later, for a slight bronchitis. He said he had gone back to work two days after going home, and had worked hard every day. Evidently his experience of three months previous had been of a purely nervous character.

Enough has been said as regards the effect of cause of the lesion on the prognosis. Before we can consistently close the subject of prognosis, it is well to mention certain personal factors that have an undoubted influence on valvular lesions. The age of a patient, for example, will affect our prognosis. Adolescence and early adult life are the ages in which these lesions offer the most favorable prognosis. As age increases the

prognosis grows more grave. The young have a better general and cardiac tone, and the heart muscle has more reserve to draw on. This makes it entirely a question of heart muscle. On the other hand the development of puberty is apt to draw heavily on the reserve and may over balance a well compensated lesion. In adult life, men are more prone to grave valvular lesions, possibly owing to the greater exposure from their occupations as well as their habits.

Pregnancy and partuition are disturbing factors in women, but are, we often have reason to believe, less serious than some authors would have us believe.

As a general rule the chances are better in those individuals of the better class, whose mode of life may be fixed by choice and not by necessity. Family history, as has been mentioned in discussing the cause of the lesion, is of importance in prognosis. A family tendency to early arterio sclerosis gives a relatively bad prognosis.

Certain associated conditions may be of great importance in the prognosis. For instance, a pericarditis associated with an endocarditis doubly affects the prognosis. As you know, we have such a condition developing say from an acute articular rheumatism. We are very apt to have a myocardial affection, thereby greatly enhancing the embarrassment of the heart as a result; compensation is broken and death may occur from heart failure within a few months of the original trouble.

Continued high arterial tension, from whatever cause, with or without sclerosis, is bad, in that the work of the heart is materially increased. We see this in all forms of Brights disease, but especially in chronic interstitial nephritis. Emphysema is also an important factor, since it raises the pulmonary pressure, thereby embarrassing the right heart. There are many other conditions productive of high blood pressure which directly or indirectly affect the prognosis of valvular lesions.

Certain accidents must always be considered in rendering a prognosis. In mitral stenosis, where the dilated auricle permits the formation of thrombi, emboli may be swept into the blood stream to be carried most often into the innominate and right carotid, finally lodging in the middle cerebral artery, producing hemiplegia or even death. Sudden death may occur from acute dilitation of the heart.

In conclusion, permit the following summary:

1. An accurate diagnosis must be made of the lesion, its extent, the condition of the heart as regard compensation, and the cause of the lesion.

2. The cause of the lesion, the age, sex, habits and family history of patient and the presence or absence of general and local disease are important in prognosis, chiefly from their effect on the heart muscle.

3. The condition of the heart muscle is the most important factor in the prognosis of valvular lesions.

FUNCTIONAL DISEASES OF THE HEART.

By T. D. Renfrow, Billings, Okla.

I suppose the reason that I chose this subject was because I did not know much about it, and, in studying the subject learn some things about the functional diseases of the heart.

In looking over the literature of this phase of heart trouble we found that the distinction between anatomical and functional diseases are not so clearly drawn as formerly.

Anatomical diseases of the heart were formerly looked upon as the only serious cardiac affections while functional disturbances were not so considered, but now, even heart death from functional disturbances is considered possible.

The failure of diastole in contrast to failure of systole in heart death leads one to consider heart death due to disorders of the nervous impulses to the heart.

Anatomical diseases of the heart may precede a functional disturbance, and, on the other hand, a functional disease end in anatomical disease.

Persons most frequently affected with diseases of the heart muscle and vascular system are those who, in early life, were the victims of neurasthenia or hysteria. We, as physicians, advise our patients to forget their self-diagnosed heart trouble because we fail to find any alterations in the size and sounds of the heart. Such advice gives little consolation to those who are the victims of some functional disturbance. We often observe the effect that severe mental strain or intense emotion has on the heart of the poor unfortunate victims of the so-called "broken heart."

The principal divisions of functional heart disturbances are divided into the affections known as tachycardia, bradycardia, cardiac arythma, angina pectoris, pseudo angin, the digestive disturbances of the heart, the affections due to sexual organs, and irritable and weak heart. While there are other classifications of functional heart diseases, I will only briefly consider the ones mentioned.

First, tachycardia, or fast heart, may occur in most all pathological processes of rapid heart, or polysystole, which increased rate is at the expense of the diastolic time in the cardiac cycle. The long pause is much shortened, but the shortened time between the systolic and diastolic sound (or the short pause of the cardiac cycle) is not apparent. By embrycardia we understand, not only an increase in the heart rate, but also a modification of the heart sounds and spacing of silent phases. In embrycardia the systolic and diastolic sounds are identical in character and there is a shortening, not only of the long pause, but the short pause as well, the two having the same length.

The distinction between polysystole and embrycardia is very clearly brought out in instances of rapid heart rate as may be produced by pres-

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(Read before the State Medical Association at Enid by T. F. Renfrow, Billings, Okla.)

sure neuritis of the vagus on which is grafted the phenomenon of paroxysmal tachycardia.

So long as the heart rate is maintained at 140 or 160 we are able to identify the phases of heart sound by their character and by the spacing of silent phases.

If an attack of paroxysmal tachycardia should appear all this differentiation is effaced and we have the phonomenon of embrycardia.

For the production of embrycardia we must have marked shortening of the refractory period of the systolic phases of the heart and shortening of the compensatory pause. Thus we see tachycardia with embrycardia exhibits phenomena which correspond to a prolonged series of extra systole.

Paroxysmal tachycardia is clinically inseparable from embrycardia. The duration of the attacks last from a few minutes to months. The onset is sudden and terminates equally as sudden. The patient will often have a premonition of impending tachycardia by occasional extra systole or intermissions. The warning lasts for half a day or five minutes. There is a feeling of oppression over the precardial area. A sense of numbness and weakness in the left arm, a feeling of sudden flow of blood to the head, and a throbbing feeling over the heart, the onset and termination is sudden if the origin is in the medulla, but not so, if in the myocardium. When vagus pressure is the cause, the rapid heart rate is constant throughout the attack.

The cause of spasmodic asthma in the child with tuberculous mediastinal glands may be regarded as a crisis of vagus disease. Cardiac crises in the course of vagus disease is a closely related conception.

I remember a patient of mine who in her usual health had a very high pulse rate, after having typhoid fever, during the convalescing period, got out on the vessel unassisted despite the warnings against exerting herself. The nurse in the adjoining room heard her getting out of bed and started to go to her assistance. The patient succeeded in getting on the vessel. The nurse saw the patient's head drop on her chest and life was ended prematurely and untimely.

Bradycardia occurs in from 4 to 17 per cent of patients. Some writers reports bradycardia as more common. The proportion of women to men all the way from 6 to 50 per cent. Bradycardia may result from reflex and direct stimulation of the vagus center from irritation of the vagus trunk or the heart itself. Bradycardia that will show increased heart rate after the administration of atropine the cause is external. Bradycardia of myocardial origin is little affected with atropine. Bradycardia which accompanies acute infection may have two sources of origin, namely, myocarditis and infection vagus neuritis.

Many of the most pronounced cases of bradycardia with a heart rate of 16 to 18 a minute have occurred in diseases of the coronary arteries. These are the conditions which are most favorable to heart block. Any process which causes an increased intra-cranial pressure may cause brady-

cardia. Bradycardia may occur in melancholia, hysteria and neurasthenia and is sometimes found in syringomyelia combined systemic diseases of the chord and tabes dorsalis. Bradycardia may be caused from pressure on the chord from neoplasms, hemorrhage, or disease of the vertebra. Bradycardia may occur in a number of intoxications, such as lead, digitalis, muscarine, picric acid, and pysostigmine. Nicotine in pharmaceutical experiment is found to slow the heart rate, but we do not see this phase of nicotine poison in smokers as the patient does not consult his physician until the latter effects of rapid heart and arythma appear. In infectious diseases several sources of bradycardia are to be considered: The infectious vagus neuritis myocarditis and toxines which affect the heart muscle. I lost a little patient from diphtheria toxine after the membrane had all disappeared. The heart had showed some weakness, but gave way suddenly with little warning. The diagnosis of bradycardia requires first to determine the relation of the arterial pulse to the number heart impulses and the number of centrifugal pulses in the veins.

To determine whether there is a genuine bradycardia, ineffectual heart beats or heart block, the procedure would be to look for evidence of disease of the brain or spinal cord, and any possible source of toxic infections or pressure of vagus neuritis, and finally we must consider the myocardia, and seek for possible sources of reflex stimuli.

Cardiac arythma is so protein in character and origin and variations in prognostic significance there is no fixed plan for interpreting its meaning. Any form of arythma must be considered in its pathological and etilogical relations before its real portent can be understood. Any disturbance of the heart rythm usually disturbs the patient, which is of an uncomfortable and sometimes terrifying nature.

The physician's diagnostic art is rarely more severely taxed than in explaining the cause and significance of some cases of arythma. It may be due to stimuli of many kinds from the meninges, cerebral cortex, base of the brain and cord. Arythma may be a symptom when there is irritation in the vast region supplied by the throracic abdominal and hypogastric sympathetic chain. And, finally, the heart itself or its direct nerve supply may be sources.

The large range of possibilities brings the clinical study of arythma in touch with the whole known and speculative field of pathological physiology.

Cardiac activity is the expression of co-ordinate excitation and inhibition in nerve paths and nerve centers, and when we consider the close anatomical relation of excitory and inhibitory paths and the centers and equilibrium of the automatic center in the heart itself with the property of the auricle and ventricle to independently assume the rythm then we have some conception of the very intricate problem.

Myocarditis may increase or diminish the heart rate or cause it to be irregular. Pressure on the vagi from tuberculous disease of media stinal

glands may cause paroxysmal bradycardia or paroxysmal tachycardia. Gaseous distention of the stomach may cause bradycardia, heart block, tachycardia, extra systole, genuine arythma, and a cycle of vagus irritation. These varying signs may appear in the same person at different times and in the same attack.

The transition from tachycardia to bradycardia with heart block has been seen. We must also consider the psychic stimuli that contribute to the production of rythm. Nervous persons occupying themselves with a study of their pulse produce arythma. Arythma in the hysterical and hypochondriacal persist constantly and for long periods of great mental or emotional distress Arythma may be paroxysmal just as rapid and slow heart assume the paroxysmal character. Irritation of the vagus trunk may cause arythma. It may accompany mediastinal disease Toxic effects on the vagi and infections and inflammations of the same. The use of digitalis and opiums in small doses, tea and coffee may cause same.

There are cases of long standing arythma which exist for years and not cause any functional impairment of the heart's work.

It would be a valuable aid if we could distinguish the various forms of arythma in their pathological significance between genuine arythma heart block, and extra systole, in which the rythm is assumed by the auricle and those which are assumed by the ventricle. But there are difficulties in the way which have not yet been solved.

Angina pectoris, a disease associated with general regional vasomotor crises and with nutritive and functional ischemia of the myocardium has received as many explanations as there are concomitant symptoms accompanying the attacks. Angina is associated with impairment of the blood irritation of the myocardium. This can arise in several ways. First, isolated disease of the coronary arteries unassociated with the disease of the aortic valves or systemic arteries. Second, disease of the coronary arteries associated with the above named. Third, partial or complete occlusion of the lumen of the coronary arteries at their origin, on account of disease the aortic valves or the root of the aorta. Fourth, vascular crises in the coronary arterial distribution, due to vasomotor influence not associated with anatomical lesions of the arteries. In order for the myocardium to meet the sudden demands made upon its reserve energy requires great fluctuations in its blood supply. This supply can only be supplied by a varying vasomotor activity. Morover, the myocardium shares with the gray matter of the central nervous system the highest degree of sensitiveness to an impaired supply of oxygen. From the above classification there is opportunity for widely differing pictures of angina. The term true and false angina are unfortunate. The patient who has symptoms of angina from vascular crises suffers from as true angina as the patient whose angina is caused from sclerosis. The difference lies in the pathological anatomy of the two kinds of angina, but the physiological pathology is the same. In both cases we are dealing with myocardial ischemia.

Patients suffering from cardio-vascular disease with attacks of angina usually present several points of tenderness to pressure.

The site of the spinal accessory as it turns over the sterno cleidomastoid muscle, the second and third ribs about one inch external to the left external border are most common sites of tenderness.

The subjective pain of angina pectoris may have a very wide distribution. The lower edge of the sternum is usually the site of the most intense pain, but it may be over the precordial area to the left of the sternum or may extend to the side of the neck or third cervical segment. In the arm the area of pain is most common in the distribution of the eighth cervical and first, second and third dorsal segments.

Epigastric pain and vomiting are common in angina from myocardial infarcts, but the gastric symptoms may be due to disease of the arterial supply to the stomach and not to the pains in the heart.

There has not been in the past sufficient appreciation of the role played by diseases of the abdominal aorta, its branches and arteries of the brain, and the accompanying vascular crises and functional and nutritive ischemia arising therefrom. Pseudo angina is a term used to describe cardiac pain unassociated with pathological lesions of the myocardia, coronary arteries, or aorta. Circulatory disorders caused by disorders of digestion are comparatively frequent. These patients are too often regarded as hypochrondriacs and neurasthenics, and that auto suggestion is the source of their trouble. They become mentally depressed and this increases their digestive disturbance and finally such patients live in constant terror.

Irritable or weak heart. Up to the present time there exists no absolute proof to my knowledge of acute passive dilitation and death from excessive muscular exercise, although there are excellent physiological and clinical reasons for believing they occur, there is a suspicion that a former infection, congenital defects or manner of living were contributory influences, although there is no doubt of the ability of a man to work a heart, which is impaired, to death. There is some doubt about a sound heart being worked to death. An athlete starts on a mile run, during the first quarter his heart beats violently and his dyspnea distresses him more than in the second quarter, he has got his second wind. This experience is due to the intervention of depressor nerve stimuli which introduce vagus influences for the conservation of the heart and vasomotor relaxation to lighten the vascular resistance. It is probably this vagus which causes the compensatory active dilitation in muscular exercise which has often been falsely interpreted as a passive dilitation and sign of the heart failing under the increased demand of physical exertion. The vagus is the diastolic and anabolic nerve of the heart and both influences are conservation measures under such circumstances. The heart is capable of greatly increasing its capacity, without causing passive dilitation, and the anabolical effects of the vagus influences the metabolism of the heart muscle in a favorable manner. The accelerator nerves are the catabolic

nerves of the heart. In paroxysmal tachycardia the heart really diminishes in size early in the attack. If the attack is prolonged for several days we then see signs of passive dilitation appear. The heart increases in size and cyanosis, dyspnea pulmonary oedema, hepatic stasis, albuminuria, oedema of the legs develop. The signs have been observed where death seemed imminent when suddenly the normal rythm of the heart was restored and all signs of incompetence promptly disappeared. Such a series of events followed by instantaneous recovery would be impossible if the tachycardia were merely a symptom of myocardial disease and not the primary affection. In such a case we are justified in saying that heart exhaustion is due purely to accelerator influences.

Experiments show that the heart's action depends upon the supply of oxygen and the removal of carbon dioxide.

Mangus showed that the mammalian heart could be maintained in rythmic contractions for an hour after the removal from the body by simply conducting a current of oxygen through the coronary arteries without any nutrient material. Furthermore, he showed that the contractions could be maintained for a short time if the products of metabolism were carried away by conducting a current of hydrogen to the coronaries. If carbonic acid gas were introduced into the coronary circulation the heart's activity promptly ceased. Precardial pain and rapid heart rate are two conspicuous symptoms in irritable heart.

The effects of tea, coffee and caffeine on the mammalian heart has been interpreted in as many ways as there are students on the subject. When coffee is drank in moderation, it may be responsible for occasional cardiac intermissions, but it is very doubtful if this can be a direct effect. It is more probable a reflex effect on the heart due to digestive disturbance. Tea and coffee certainly favor the production of paroxysmal tachycardia in patients who have such attacks secondarily to gaseous distensions of the stomach.

Secondary to digestive defects are the modifications of blood distribution. Patients with gastro-intestinal dyspepsia and ptosis of stomach are troubled with cold hands and feet and have a radial pulse of small volume and low pressure. Some people become chilly after eating their meals. The circulatory disturbances associated with digestive disturbances are of a mild type. While with ulcer and cancer of the stomach, obstruction of the bowels, peritonitis, ascites, and metearism, are never associated with such cardio vascular symptoms, which seems strange.

Explanatory of the disparity, it has been suggested that grave diseases depress the reflex centers, whereas, the milder affections are associated with excitation.

While there are several other subdivisions of the functional diseases of the heart I will conclude my subject with a slight consideration of the influence of the sexual organs on the heart.

The pelvic organs and genitalia have a rich sympathetic nerve supply. From the inferior hypo-gastric plexus, which is a continuation of the solar

and aortic plexuses there is also a direct connection between the renal plexus and the ovaries. Vasomotor depression and accelerator impulses to the heart are common manifestations associated with irritation of the pelvic organs in both male and female. Cardiac palpitation with rapid pulse and slightly increased cardiac volume are described as results of excessive masturbation. Probably the cerebral cortex in such cases is as much a source of these symptoms as the sexual organs. Any factor which is so productive of painful mental experiences and introspection cannot be clearly differentiated as the sole cause of disturbance in the rate and rythm of the heart. The beginning and end of menstruation are often associated with palpitation, rapid heart and sudden vasomotor oscillations, hot flushes, palpitation, and rapid heart are symptoms most women expect at the menopause. The female insane have exacerbations of cerebral symptoms at their menstrual periods.

SURGERY OF THE HEART.

Dr. R. M. Howard, Oklahoma City, Okla.

Surgery of the heart is a subject comparatively recent. Fifteen years ago the heart had not been brought within the surgeon's field, and wounds of the heart particularly were beyond surgical treatment. One author in 1895 described the treatment of wounds of the heart as follows: Lowering the head to prevent cerebral anemia, the administration of opium to relieve pain and to control inflammation, and the application of artificial warmth to the surface of the body. In 1896 Farina reported the first recorded case of suture of the heart wall for a penetrating wound, and, while his patient died on the fifth day from penumonia, the operation seems to have been successful as far as repair of the damaged heart was concerned. Operations on the pericardium, however, were done as early as a century ago and more. In the memoirs of Baron Larrey we read that that distinguished Frenchman aspirated the pericardial sac in 1798, and we see reference to this phase of heart surgery throughout the literature of the past century. But even this operation was not done extensively until within the past few years. Wounds of the heart are not necessarily fatal, as popular notion would indicate. Writers from the time of Pare down to our own time relate cases of persons living for a longer or shorter time after heart injury. G. Fisher in the middle of the last century compiled 452 cases of heart wounds and found that from 7 to 10 per cent recovered completely. Even rupture of the heart, due to some pathological condition, is not always immediately fatal, Mumford citing a case of this type that survived for nine days. Degeneration of the heart muscle, however, renders any attempt at repair futile in these cases. The majority of persons receiving injury of the heart—even perforating wounds—do not die directly from the injury but from the subsequent loss of blood combined with the emptying of the heart, the result of pressure of the extravasated blood on the large veins. It has been proven by statistics that a perforating wound will heal firmly if the results of bleeding are

avoided. A fibrous scar is formed, however, which may later lead to the formation of an aneurism and rupture. Practical surgery of the heart is confined at the present time to operations for heart wounds, operations on the pericardium and operations for adhesions between the heart and parietal pericardium, or chest wall. Paracentesis Auriculi has been suggested, but until further experimental work has been done along this line we are not justified in attempting it.

Heart wounds produce instant and alarming symptoms. Pain, hemorrhage—usually free, sometimes slight—palpitation, syncope and dyspnoea. The symptoms depend on the location and extent of the injury. Death is instantaneous if the ventricle is torn widely, or the center for heart block is damaged, or if the auricles are injured. Fortunately the ventricle is injured most commonly, the left more often than the right. The wall of the ventricle may be injured without perforation. This superficial wound may bleed profusely, thus confusing the diagnosis. A perforating wound if small may bleed but little, owing to its being closed by the interlocking of the heart muscle with every systole. An important complication of these wounds may be coincident damage to the lung and pleura. The surgeon must take note of those victims of heart wounds who do not die immediately. These are the cases where surgery is important. If a patient who has received an injury in the region of the heart is found suffering from great dyspnoea with cyanosis, or from collapse and anaemia, and with a corresponding variation of the pulse, and if on examination hemorrhage into the pericardium, with or without hemmothorax, is discovered immediate exposure of the heart is clearly indicated; first, with the object of opening the pericardium and emptying out the extravasated blood which is compressing the heart, and second, of suturing the wound in the heart. This should be done under the best aseptic technic attainable, and with the least possible damage to the surrounding structures. This treatment should be supplemented by proper stimulation—hot bottles, raising the foot of the bed, a hypodermic of morphine and atropine, the intravenous injection of normal salt solution with adrenalin 1 to 50,000, and the application of the pneumatic suit of Crile, if it is available. It is rarely necessary in these cases to give an anesthetic, but if one is used, ether only is permissible. This character of work, as well as all the work about the chest, should be done under differential pressure if possible. It is hardly necessary here to go into the steps of the operation, as some ten or twelve methods have been advocated. The method of Kocher is admirably described in our latest text books and it is the one most used. The following procedure, however, is common to all. With the patient in the Trendelenburg position cleanse the skin thoroughly, enlarge the external wound and ascertain the condition of the underlying cartilages and ribs. If they are found divided advance through the opening thus provided. If they are intact, turn back a rib or several ribs—first, the sixth and up as needed. Seek and tampon any rent in the pleura; expose the pericardium and find the wound in that membrane; enlarge the pericardial wound emptying the

pericardium of blood and clots, and look for the wound in the heart. Having found the wound suture it, using a round intestinal needle, with a continuous suture of cat gut or silk, being careful not to puncture the endocardium. Sponge out the pericardium and suture it, providing for the drainage. Suture any rent in the pleura and drain this area. The internal mammary vessels should always be inspected for any possible damage. Certain wounds of the heart offer brilliant opportunities for the surgeon. Without operation 90 per cent die; with operation, 64 per cent and the mortality is falling. Gun shot wounds are more fatal than stab wounds, but even they may sometimes be repaired.

Pericardial effusions, especially the simpler forms, may be dealt with by aspiration and incision. In such cases aspiration is comparatively easy and safe because the heart is crowded back into the depth of the pericardium. Aspiration is not safe, however, in cases of purulent effusion because the heart apex may be held forward by adhesions. The base of the pericardium extends transversely across the roof of the ensiform process from a point three-fourths of an inch to the right of the sternum to a point three inches to the left of the sternum. Above, it reaches as high as the second rib; its posterior surface reaches as high as a line drawn through the middle of the manubrium sterni.

Paracentisis of the pericardium is indicated according to Curschman in cases of serous, sero fibrinous and hemorrhagic effusions. First, when the fluid is rapidly increasing and oppressing the heart and lungs, causing dyspnoea cyanosis and a small rapid pulse. And second, when absorption does not take place, especially if there is concurrent disease of the heart and lungs, or the presence of fluid in the pleura. In selecting the site where we should puncture, which is only indicated where there is an extensive effusion, Curschman has shown that the choice is determined by conditions quite different from those which fix the site when the heart itself is to be reached in cases of injury to that organ. The portion of the pericardium which is closest in contact with the thoracic wall is to be avoided in performing paracentisis for when fluid accumulates in the pericardium it does so chiefly laterally and at the same time distends the pericardium outward, especially to the left and backward. In this way the heart itself comes close to the chest wall. Curschman, therefore, advises puncture in the mammary line in the fifth and sixth intercostal space. The needle, of course, traverses both layers of the pleura, although the lung escapes as it is pushed aside in all extensive effusions, which alone justify puncture. If puncture is made close to the sternum care must be exercised to avoid the internal mammary vessels, also direct injury to the heart. The use of the puncture in small effusions should be very much limited.

When the effusion is sero fibrinous, or hemorrhagic in addition to causing pressure on the heart there is danger of pericardial adhesions. These are best prevented by pericardiotomy. In suppurative pericarditis simple puncture is no longer regarded as adequate. Statistics show that

in nine cases of suppurative pericarditis treated by puncture all died. On the other hand, there were six recoveries in nineteen cases treated by incision without resection, and eight recoveries in fourteen cases treated by incision and resection. It has been shown by experiments with gelatin injections that in a sitting posture small effusions collect between the anterior attachment of the diaphragm and toward the apex, so this is considered the best place to secure suitable drainage. Resection of a rib and opening of the pericardium may be done under local anesthesia if necessary. There seems to be no reason why the flap operation used in exposing the heart in injury cases should not be used in pericardial effusions as it gives the best and safest exposure.

As a result of injury or of the irritation of a pericarditis we may get more or less, sometimes extensive adhesions between the heart and parietal pericardium or chest wall, with the resulting symptoms of pain, palpitation, dyspnoea, cyanosis, congestion and ascites. For the relief of these symptoms the pericardium has been opened and the adhesions broken up. This operation is termed cardiolytis; or the chest wall may be imobilized by resection of ribs and the sternum, one or both. Simply cutting the costal cartilages may give relief. Delorme devised this type of operation originally for the relief of pleural adhesions and later applied it to the heart In these cases it is probably best to resect the periosteum to prevent the formation of new bone. To prevent the formation of dense adhesions between the heart and pericardium in suppurative pericarditis it is advisable to interfere at an early stage and wash out the coagulated fibrin. Normal salt solution is the best fluid to use for this purpose. Mumford in his latest book says:

"In recent years the possibilities of heart surgery, or direct operation dealing with the heart, have been made to appear as important future possibilities. George W. Crile in 1903-4 published an extremely interesting series of experiments and operations on the heart showing that after apparent death in dogs and man, even when a half hour of suspended animation has elapsed, the heart may be stimulated to resume its function by direct rhythmic pressure over the pericardium, or by sub-diaphragmatic massage—the abdomen being opened for the purpose—supplemented by artificial respiration and long-continued infusion of 1 to 50,000 adrenelin solution.

Harvey Cushing working in the Huntarian laboratory of Johns Hopkins University has demonstrated the possibility of producing artificial cardiac lesions by intraventricular incision of the cardiac valves with a resulting recovery from the operation except for the cardiac lesion. This extremely interesting work suggests the possibility of intra-cardiac manipulation for the relief of valvular stenosis.

Theoretic as these considerations may be, the work of such experimenters has proved conclusively that the heart may be approached and handled with boldness. At the same time the practical experience of many

surgeons has shown the reasonableness and importance of operating upon the heart and pericardium for traumatic lesions of these structures. In all of this we are considering a strikingly interesting and little explored field of surgery."

ARTERIO SCLEROSIS WITH SPECIAL REFERENCE TO SYMPTOMS AND PHYSICAL SIGNS.

By L. J. Moorman, M. D., Oklahoma City.

In 1911 Hertz sent out question blanks to physicians in Austria, asking an opinion regarding the factors involved in the production of arterio sclerosis. He received 822 answers and less than one-fourth of them agreed upon any one influence as the main factor. Among the 822 opinions, eleven different influences were suggested as the chief cause.

W. S. Thayer says, "Great as is the importance of arterial changes in relation to many of the ills to which flesh is heir and numerous as have been the anatomical clinical and experimental researches concerning this subject, it must be acknowledged that there is much yet to be learned with regard to the etiology, the manner of development, the nature of the changes in the different parts of the arterial tree, their relation to variations in blood pressure and to visceral disease, as well as concerning the relations of peripheral to central changes; and there are still wide differences of opinion as to the interpretation of some of the observations which have been made."

Quoting Osler, "Discussion is still active on the finer changes in the small vessels in arterio sclerosis. The controversies of the seventies, started by George Johnson, Gull and Sutton, Dickenson and others still rage. Where does the process begin? Is there a true hypertrophy of the muscle fibers? What is the relation of high tension to the sclerosis? Which comes first? Is the primary mischief caused by the action of a toxine in the finer tissues of the capillaries and arteries? Or do these irritating substances cause spasm of the smaller vessels and so raise the tension? What is the relation of the involuntary changes in the vessels in old age to those met with in younger persons? May not increased viscosity of the blood play an important role in causing high tension and arterial strain? We cannot say that any of these problems are finally settled, and the whole question is in the melting pot again in consequence of the remarkable studies on exeprimental arterio sclerosis. We lack definite knowledge of the finer changes in the capillaries, which are probably always involved, and through which, after all, the essential processes of the circulation are carried on."

It is unfortunate that so little is definitely known about a disease so common and so relentless in its toll of human life. Elsner says, "Arterial changes furnish the fundamental morbid processes in the largest number of deaths after forty." Osler has aptly expressed in the following words a truth conceded by all, "The tragedies of life are largely arterial."

Without further reference to these questions still under controversy, we pass to the consideration of symptoms and physical signs. Arterio-sclerosis may disturb the function of an organ:

1st. By reducing its capacity for work through limitation of its blood supply.

2nd. By necrosis or gangrene through obliteration of vessels where collateral circulation is impossible.

3rd. By vascular spasm, through increased irritability of the smaller vessels.

When the term arterio-sclerosis is employed, no doubt most of us think of a rather corpulent man about fifty-five who has led an active business life, whose exercise consists of his daily trips to and from his office and these perhaps in an automobile or street car with the daily paper in hand; a man who has eaten three hearty meals a day, whose amusements have been confined to social intercourse with friends of like tendencies. At this age he is beginning to experience unpleasant sensations, such as unusual shortness of breath when he runs for his car, a dizzy sensation when he stoops to lace his shoe; he may notice some palpitation and an uneasy feeling about the heart and at the end of the day he may notice some swelling of the ankles. Upon examination, the heart shows slight enlargement, the apex impulse increased and displaced to the left, the pulse is not easily compressed, the radial may be palpated after the pulse is obliterated, the first sound of the heart is prolonged, the second aortic sound is accentuated. The urine may or may not show evidence of kidney change. However, it must not be forgotten that practically the same conditions may be found in a man who has led a very temperate life and whose business has been seasoned with healthy recreation. The same objective signs may be discovered by accident in an individual who has suffered no inconvenience or discomfort. In the study of arterio-sclerosis, one must ever keep in mind its varied manifestations, its wide range of severity, and its possible occurrence in all classes, from infancy to old age.

Thayer has warned us against the tendency to attribute obscure symptoms to arterio-sclerosis merely because of the presence of palpable radials and tortuous temporals. Here, also, it is well to remark, that the absence of demonstrable peripheral arterio-sclerosis does not preclude the possibility of advanced sclerosis of deeper vessels. Under special manifestations we may consider the following:

1st. Nervous system.
2nd. Cardiac.
3rd. Renal.
4th. Abdominal.
5th. Peripheral.
6th. Pulmonary.
7th. Uterine.

1st. Nervous system. The individual suffering from arteriosclerosis may early present a train of symptoms corresponding to neurasthenia. Headache

may be an early symptom, usually associated with high tension and relieved if the tension is reduced. Vertigo is a common symptom and may be excited by exertion or by certain positions of the body. There may be hemiplegia, monoplegia, paraplegia and aphasia, all disappearing in a short time if due to vascular spasm or persisting if due to rupture of the vessel. The greatest danger is from rupture of cerebral vessels with resulting apoplexy or the gradual occlusion of cerebral arteries with necrosis and softening of brain cells and progressive failure of mental powers leading to dementia. Cases of arterio-sclerosis, involving the vessels of the spinal cord have been reported with progressive paraplegia, incontinance of urine and feces, loss of knee jerk, etc. Epileptiform convulsions may occur. Such convulsions developing after middle life should always cause one to suspect arterio-sclerosis. True epilepsy usually develops in early life.

2nd. Cardiac. Valvular and myocardial lesions may result from arterio-sclerosis. Many cases of aortic insufficiency are directly due to sclerotic changes in aorta and valves. The same process may cause incompetency of the mitral valves. The coronary vessels often show advanced sclerosis with narrowed lumen, limiting the blood supply to the myocardium. The heart muscle, already hypertrophied on account of peripheral resistance, begins to show weakness with degeneration. A break in compensation with all the sypmtoms which accompany dilitation may follow any increased demand upon the weakened heart muscle. Branches of the coronaries may be obliterated by endarteritis or by blocking with embolus, or sudden narrowing may be caused by vascular spasm. All these latter conditions may give rise to distressing attacks of angina pectoris, often proving fatal. If the sclerotic process should involve, either directly or indirectly, the auriculo-ventricular bundle we may have irregularities or even the Stokes-Adams syndrome.

3rd. Renal. The urine may be increased in quantity with low specific gravity, albumen absent or in small amount with hyalin casts and often red blood cells, the small contracted kidney, or it may be normal in quantity with normal or high specific gravity and no albumen. Later albumen may be abundant with numerous casts—arteriosclerotic kidney.

4th. Abdominal. Some one has said, "Happy is he whose splanchnics are normal and are offering no resistance to the circulation." It is believed that arteriosclerosis of these vessels almost invariably causes high tension. There may be a sense of weight or oppression in the epigastrium. Gastric ulcer may be simulated and differentiated only after careful study and continued observation. This condition of the splanchnics may be marked by recurrent attacks of abdominal pain with or without vomiting. These attacks, supposedly due to vascular spasm, may be mistaken for hepatic colic or other abdominal pains.

5th. Peripheral. It is not uncommon to see, as a result of endarteritis obliterous, dry gangrene of a toe or finger or even the whole foot or hand. This gradual cutting off of blood supply is not, as a rule, painful.

One of the most frequent results of arterio-sclerosis of the extremities is the condition known as intermittent claudication. A cramping pain with loss of power brought about by the inability of stiffened and narrowed arteries to meet the demands of an active muscle. Rest promptly restores function and comfort. The inspection and palpation of peripheral vessels, together with the opthalmoscopic examination and the estimation of blood pressure furnish much valuable information.

6th. Pulmonary. The pulmonary artery may participate in a general arteriosclerosis with physical signs of involvment of the main trunk of the artery and enlargement of the right heart. There is early cyanosis without marked dyspnoea or edema. Repeated hemorrhages from the lungs may occur.

7th. Uterine. Arteriosclerosis of the uterus seems to predispose to uterine hemorrhage. This may manifest itself as menorrhagia or metorrhagia. There is a difference of opinion as to the direct cause of this excessive bleeding, but in all probability it is due to improper muscular contraction resulting from imperfect blood supply.

DISCUSSION.

Dr. White:

A symposium on these subjects necessarily cannot cover the subject in detail, and yet it seems to me that these papers, so far as their titles are concerned have done well. The paper of Dr. Howard interested me very much. Being a little out of my line, and having had no work at all as to the surgery of the heart I do not feel competent to discuss it.

The paper of Dr. Moorman was of special interest and appears to me as being a most excellent one. He lays special stress on the early hardening of the arteries. I think we overlook that. We look for the change in the kidney first thing and often we find that before the arteries manifest any special change, but many times find the hardening of the arteries giving rise to disturbances of the liver and stomach and so on. I do not like to consider arteriosclerosis as a disease. It is a part of the expected change of nature. We have a case on record of a man that lived one hundred and fifty-two years and died with no evidence of arteriosclerosis, but that fact does not change the thing at all to my mind. It is a question of how long a man lives whether he will develop arteriosclerosis, and to figure it as a disease I do not think is right. I think the same thing might be applied to the paper as to title, "functional diseases of the heart." The proposition is that I question almost whether we have functional diseases of the heart. As to diseases, I do not believe they exist per se in the heart. A disease in some other part of the body will change the action of the heart, but it is a manifestation of the other trouble; or trouble elsewhere.

Dr. A. P. Presson, Kingfisher:

One state of heart lesion has been brought to my attention within

the last year. I was in consultation with another doctor and did not find anything except a bare murmur and the symptoms of uncompensated mitral sclerosis. They sent for me one night, saying he was having one of his conditions of spasmodic trouble. I put my ear to the chest and listened and I heard this: * * * * * With this clue we found what we did not know before. The man died some weeks afterwards. I had one case of nitral uncompensated. The patient went to California about a year ago. I did not think he would live to get there. He weighed at the time he left two hundred and seventy pounds. He went to Long Beach, California, without any change of treatment that I could see and in less than three months time he lost ninety pounds, and is living there today in reasonable comfort. The only thing that made the change was the going from this climate to that one, and I think he will live for several years, and he could not have lived through the summer if he had stayed here.

Dr. L. A. Riley, Oklahoma City:

This symposium on diseases of the heart and blood vessels is to my notion a very excellent way of getting at these subjects, from the medical side as well as the surgical side. The question of arteriosclerosis in connection with diseases of the heart is one that is consuming more study at the present time than it ever has before. I suppose no cause is more discussed or sought for than the cause of the early prevalence of it, by the most accurate diagnosis under the most accurate mechanical devices.

Arteriosclerosis may be either general or local. Localized arteriosclerosis may occur in any particular part of the anatomy. It may even be in the capillaries; they occasion more resistance than the larger vessels. If one can go past the capillaries into the veins, venous sclerosis is something we know but little about at the present time, but I think it will be developed in the future. It has been generally considered that arteriosclerosis is caused by high blood pressure. That is not necessarily the case. I do not believe the etiology of arteriosclerosis has been very fully emphasized yet, but I believe there must be a primary condition of the arteries which we inherit which makes them more susceptible to become in this condition in some people more than in others, which in some cases makes the arteriosclerosis occur in young people. Of course, we expect any man living long enough will have arteriosclerosis, but those developing in early life must have inherited a deficient condition of the arterial walls, which are more easily irritated by intoxicants, such as alcohol, or typhoid fever or pneumonia or any condition that places an irritative condition on arterial walls. I think the subject of arteriosclerosis is in its infancy now and the high tension under which people are living now may make it more prevalent, and again it may not.

The Chairman:

If there is no further discussion we will call on Dr. Williams. If there is no further discussion by the doctor we will call on Dr. Renfrow.

Dr. Williams:

I came in late, and if there was any discussion on my paper I did not hear it. If there was, I thank you for it.

The Chairman: Dr. Howard?

Dr. Howard: I want to thank the society for receiving my paper and listening to it as nicely as they did.

The Chairman: Dr. Moorman?

Dr. Moorman: I just came in and heard only the discussion of Dr. Riley. I want to thank the society for the opportunity to read the paper and want to state that the symptoms and signs which I tried to bring out and emphasize are those which you get in well-developed cases of arteriosclerosis. I think we should be on the alert for the beginning of arteriosclerosis and especially for those cases of high tension where it is impossible to put your finger on the cause at the time, and by studying those cases we may find something in the daily habits of the individual which may be corrected or some condition which if watched we may be able to stay for a time, at least, the ravages that usually follow. I thank you.

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Local news of possible interest to the medical profession, notes on removals, changes in address, deaths and weddings will be gratefully received.

Advertising of articles, drugs or compounds not approved by the Council on Pharmacy of the A. M. A. will not be accepted.

Advertising rates will be supplied on application. It is suggested that wherever possible members of the State Association should patronize our advertisers in preference to others as a matter of fair reciprocity.

EDITORIAL

A CONTRAST IN MEDICAL PUBLICATIONS.

Last month we had the pleasure of calling attention to the high class of the Journal of the Southern Medical Association and its advertising pages.

We regret to have to note a contrast on analyzing a journal familiar to many Oklahoma readers. The Medical Herald, the official organ of the Medical Association of the Southwest, has long been an object of comment for its laxness in the acceptance of questionable advertising. That this laxness will eventually build up a defense for inferior products and render the physician users of them callous along ethical lines, is evident from the tenor of editorials in this publication.

The August issue of the Journal contains a criticism of the Carnegie foundation and the A. M. A. in their efforts to improve the condition of our medical colleges.

The writer of this editorial, Dr. T. D. Crothers of Hartford, Conn., a contributing editor, bemoans the fact that the medical colleges of the United States have been reduced to 117; that it is proposed to make a

reduction of 25 more. Dr. Crothers objects to reports of inefficiency of medical institutions, but suggests no remedy.

In the September issue of his Journal, he objects to the method of publication of papers in the Journal of the A. M. A., and intimates that there is a trust or combination for the purpose of limiting the output of that Journal to those who are popular with the section officers.

It is a notorious fact that preachers often overlook their greatest field of activity in their own immediate circle. That reformers overlook in their own neighborhood a fertile field for work, and in this connection we have only to call attention of this able man to his own medical publication and incidentally, we desire also to call the attention of the Oklahoma readers to the advertising columns of this journal and have to suggest that they use their influence to reform its advertising pages.

The August issue contains the advertisements of 64 drugs or compounds; of the 64, 55 have either not been accepted by the Council on Pharmacy and Chemistry of the A. M. A., have been thoroughly tested and failed, or found wanting in various degrees.

Among the oldest offenders, we find Anasarcin, Cactina Pellets, Hayden's Viburnum Compound, Triacol, Farwell & Rhines Gluten Flour, Pepto-Mangan, Gude, Nephritin, Sinkina and many others.

Dr. Crothers is an Alienist and Neurologist, we understand, of some note; we wonder how much "Neurilla" manufactured by the Dad Chemical Company of New York, he uses in his practice? How much "Neurosine" he orders dosed out in his Hartford hospital? An inspection of the advertising pages of this journal with which he is connected will convince anyone that it is a most notorious offender against ethics in carrying these advertisements to the medical profession existing today in this country. We note advertisement of compounds that were guilty of various shortcomings more than ten years ago, and that have not improved in that time. A great many of the 55 undesirable compounds have been criticised either by direct report of the Council or editorially in the Journal of the A. M. A. more than once. Most of them are known to be rank frauds, others claimers of virtues that never existed, the remainder just indifferent products.

Just as long as the unthinking physician supports these medical publications by subscribing for them, or using the products advertised in them, he will continue to do himself an injustice, and ofttimes a severe one.

We prayerfully commend the advertising pages of the Medical Herald to Dr. Crothers and suggest that there his explosive activity will find something worth working on.

ACTION OF TUBERCULOSIS.

The National Association for the study and prevention of tuberculosis have issued a statement of the activity for various State Legislatures with reference to the prevention and treatment of tuberculosis.

The statement shows that forty-one State Legislatures enacted laws dealing with tuberculosis, thirty-four states considered the prevention of the disease. During the year 1913, more than five million dollars was set aside on account of the prevention of tuberculosis, most of which was for the maintenance of state institutions.

There are at this time thirty-nine state institutions in thirty-one states. The appropriations on this account by Congress will reach one million dollars, this going to the maintenance of hospitals of the United States Public Health Service, the Army and Navy and the tubercular hospital of the District of Columbia.

Colorado passed an act requiring registration of tubercular cases. Minnesota and Wisconsin acts, subsidizing local hospitals. Indiana, creating county hospitals, while Ohio and California established bureaus for the prevention of tuberculosis.

DOING SURGERY WITH A THERAPEUTIC LAMP.

We have about decided that criticism of Osteopaths, Christian Scientists and Faith Healers is all out of place in medical journals until we have placed some of our own writers where they belong.

Every well balanced physician knows that certain conditions are surgical and practically amenable to surgery only. The fact that a ruptured gall-bladder or appendicitis breaks its way into the intestine and drains the patient to good health should not lead a physician to expect them all to do it. The subsidence of an inflammatory condition, which usually requires surgical relief, does not justify us to trust that rare occurrence to happen.

A writer in the "Hahemannian Monthly" for July, 1913, collects and publishes with great gravity the number of cures of surgical conditions with the therapeutic lamp. Case after case of appendicitis is reported as cured and he concludes that the majority of cases of appendicitis, where there is no foreign substance or pus has not formed, can be cured with a therapeutic lamp. Just how many he would kill, he does not estimate.

The writer flutes up and down the field of infection from appendicitis, abscessed teeth, through blood poisoning, erysipelas, and various others, down to septicaemia and treats them all with his lamp. Of course, not so many will accept this doctrine with any seriousness, but it might fall occasionally on fertile soil and do the patient and the physician great harm.

We believe that a bag of ice placed over the appendix by the Christian Scientist will be found more efficacious than the use of this lamp in such cases by a physician, and neither of them are to be considered as having much ultimate effect on the condition which is to be relieved properly by operation only, as a general rule.

PERSONAL AND GENERAL NEWS

Press reports state that Dr. W. C. Graves of McAlester recently suffered from a stroke of paralysis.

Dr. Claude Chambers of Claremore has been appointed physician in the Indian Medical Service. He is stationed at Tahatchie, N. M.

Dr. S. N. Mayberry of Enid spent August at the Mayo Clinic.

Dr. F. B. Fite, Muskogee, attended the International Hygienic Conference in Buffalo as a delegate from Oklahoma.

Dr. J. W. Hoover of Sapulpa and Mrs. K. Lyford were married in Kansas City September 10th.

Dr. Joseph E. Brookshire of Nowata and Miss Anna C. Steward were married in Colorado Springs August 23rd. They will make their future home in Nowata.

Dr. Leigh F. Watson of Oklahoma City and Miss Dora Lowe Watson were married September 15th at Fairmount, West Virginia. They will be at home in Oklahoma City after October first.

McIntosh County Medical Society met in Checotah September 9th. The Society presented a clinic of six cases of pellagra. Dr. J. C. Watkins read a paper on Pellagra which was discussed at length. The visiting physicians were Drs White, Montgomery, Newton and Thompson of Muskogee. The Society entertained the visitors with a dinner at the Gentry Hotel.

Dr. Geo. A. Nieman of Ponca City and Miss Grace Taliaferro of Atchison, Kansas, were recently married.

NORTHEAST OKLAHOMA MEDICAL SOCIETY PROGRAM

Bartlesville, Tuesday, October 14, 1913.

Vaccines in Surgery, C. M. Ament, Sapulpa.

Incontinence in the Female—Prognosis and Treatment, G. A. Wall, Oklahoma City.

One Thousand Cases of Labor, P. H. Mayginnis, Tulsa.

Retrospect of Sixteen Hundred Labor Cases, J. Donohoo, Afton.

Care of the Eye in the Newly-born, W. A. Cook, Tulsa.

Intratracheal Anesthesia—Demonstration on Dog, B. W. Freer, Nowata.

Phylacogenin Rheumatism, C. W. Beson, Claremore.

Bronchial Asthma and Its Treatment, R. A. Douglas, Collinsville.

REPORT OF EXAMINATION HELD AT GUTHRIE, JULY 8-10, 1913, BY OKLAHOMA STATE BOARD OF MEDICAL EXAMINERS.

Albert D. Wehinger	Texas Christian University	1913	80
Joseph C. Dunn	Eclectic M. C., Cincinnati	1897	82
Casper A. Hicks	Memphis Hospital Medical College	1913	81
Robt. E. Jamison	Marquette University	1912	75
Henry F. Knabb	St. Louis University	1911	77
Harry A. K. Hill	Bennett	1913	81
Owen K. Haydon	Kansas Medical College	1913	84
Everard B. Hamilton	Tulane	1913	85
Joseph Edward Gray	University Medical College, Kansas City	1913	85
Oscar R. Crogan	Texas Christian University	1913	82
Albert N. Earnest	Memphis Hospital Medical College	1913	80
May Adah Drew	University of Oklahoma	1913	87
Geo. Henry Clulow	Western Reserve	1912	84
Daniel W. Carroll	Vanderbilt	1913	79
Charles B. Barker	Chicago College of M. & S.	1912	88
Alvin Ray Wiley	University of Oklahoma	1913	86
Asa Wright	University of Oklahoma	1913	81
Wesley I. Wimberly	Tulane	1913	84
Vernon L. McPherson	University of Arkansas	1913	76
E. Catherine Threlkeld	American Medical College	1913	86
Joseph P. Lee	St. Louis College P. & S.	1913	79
Thomas LeRoy Lauderdale	University of Oklahoma	1913	84
Elmer Allen Leisure	Chicago College M. & S.	1913	90
Wilson Hewette Lane	Chicago College M. & S.	1913	84
Loyal M. Martin	Rush	1913	87
Elam J. Nienstedt	American Medical College	1913	85
Harry P. Price	University of Oklahoma	1913	85
James E. Parramore	Memphis Hospital Medical College	1913	86
James Willson Rollo	University of Oklahoma	1913	84
Robert H. Riley	University of Oklahoma	1913	89
Harry James Sims	University of Tennessee	1913	83
R. Earle Smith	University of Oklahoma	1913	82
P. Franklin Smith	Meharry Medical College	1913	82
Isaac Walton Rogers	Chattanooga Medical College	1906	70

The following failed to make a passing grade:

	Memphis Hospital Medical College	1911	66

The following have been licensed by reciprocity since the last report:

John B. Chapman	Kentucky School of Medicine	1877	Texas
Salathiel L. Hollingsworth	Tulane	1905	Miss.
Geo. Oscar Marsh	Tulane	1895	Ark.
Henry S. Drummond	Vanderbilt	1912	Ark.
Francis Taylor Isbell	Memphis Hospital Medical College	1901	Ark.
Thomas J. Stout	University of Nashville	1901	Ark.
Clarence B. Billingsley	Memphis Hospital Medical College	1910	Miss.
Fannie Stone-English	American School of Osteo.	1911	Mo.
Charlie Huse Hale	Kentucky School of Medicine	1908	N. Mex.
Marion Albert Clark	Meharry	1910	Tenn.
Edward Forrest Ellis	Missouri Medical College	1885	Ark.
Charles Frazier Clark	University of Illinois	1907	Neb.
Wm. Patrick Lipscomb	Vanderbilt	1911	Tenn.
Turner Fred Roberts	University of Louisville	1898	Texas
Archie Bee	Starling	1890	W. Va.
Lawrence H. Hill	Washington University	1907	Ark.
James L. Colquit	Vanderbilt	1887	Texas
Roscoe M. Jernigan	American Medical College (Ec.)	1912	Ark.
Claude Edward Laws	Chicago College M. & S. (Ec.)	1903	Ark.
Isaac H. LaMar	Ec. Medical College, Cincinnati	1886	Texas
Francis Dameron Garrett	American School of Osteo.	1909	Mo.
Novel Walter Campbell	Kentucky School of Medicine	1894	Texas
Bruce Younger	University of Louisville	1909	N. Mex.

NEW BOOKS

SURGICAL CLINICS OF JOHN B. MURPHY, M. D.

Volume II, Number IV. (August, 1913.)

The Surgical Clinics of John B. Murphy, M. D., at Mercy Hospital, Chicago, Volume II, Number IV. (August, 1913.) Octavo of 206 pages, 49 illustrations. Philadelphia and London: W. B. Saunders Company, 1913. Published Bi-Monthly. Price per year: Paper, $8; Cloth, $12.

This number of Murphy's Clinics is believed extremely interesting. Notwithstanding the fact that there is a great deal of repetition in his article on "Vaccine and Serum Theraphy" it is interesting and instructive. Wthout going into labatory detail it gives one a clear and concise idea of these preparatons and methods of admnistration.

The skiagraph showing the blood supply of various joints is a work of art and commendable. In his lecture on the care of patients after spinal injuries he emphasizes the trophic disturbances which accompany lesion of this character and admonishes the attending physician to refrain from the use of the catheter due to the fact that cystitis and possibly more serious complications invariably follow such action. The reported joint cases are similar to many he has described in prevous numbers, but his dealing with the bone cyst of the radius is rather unique and a point which should prove of great value to the general practitioner or surgeon who has not that advantage of the large amount of hospital work enjoyed by surgeons in the larger cities.

MARRIAGE AND GENETICS.

Laws of Human Breeding and Applied Eugenics. By Charles A. L. Reed, M. D., F. C. S. pp. 182. (5 1-4x7 1-4.) Price, including Postage, $1. Subscription only. The Galton Press, Publishers, Cincinnati, Ohio.

This is a stirring, practical and comprehensive appeal to the human race to improve itself by proper breeding. It is an appeal by one of the well known and able men of the profession to his fellows to interest themselves in proper marriage and genetics. An appeal to interdict the marriage of the syphilitic, gonorrhoeic, tuberculous, epileptic and other reducers of the high standard of mankind.

It is full of scientific facts relative to mis-mating and the quotations of authorities on such subjects are numerous and convincing.

When mankind thinks of these things they will devise remedies, the task we have to accomplish is to point out to them the dangers of the situation and awaken them to a realization of their responsibilities.

The author, his book and the subject need no introduction to many of the profession, but to others they do. It is sufficient to say that every true physician should read this book and co-operate with teachers, ministers and humanitarians generally in a wide propaganda of the subject.

Malaria, Etiology, Pathology, Diagnosis, Prophylaxis and Treatment. By Graham E. Henson, M. D., Member of the American Medical Association, Florida Medical Association, Southern Medical Association, American Society of Tropical Medicine, Medical Reserve Corps, United States Army (Non-Active List) with introduction by Charles C. B. Bass, M. D., Professor of Experimental Medicine, Medical Department Tulane University, New Orleans. Illustrated, Cloth, 190 pages, price $2.50, C. V. Mosby Company, St. Louis, 1913.

This is an able monograph on the subject of malaria in its many phases. Aside from the careful handling of the questions of etiology, pathology, prophylaxis and symptomatology the book is especially valuable in its suggestions as to treatment Those who have had experience in the treatment of obstinate malarial infections will readily agree with the author in his suggestions as to the hypodermatic use of quinine salts as being the mode of superiority especially in intractable and pernicious types.

The Propaganda for Reform in Property Medicines, from the press of the American Medical Association, Chicago, Ill. Paper, 277 pages, price 15 cents. Cloth 25c.

This little volume is the result of the recent activity of the Council on Pharmacy and Chemistry of the A. M. A. and is the 7th edition of a similar work. We note that especial attention is given to the claims of the exploiters of Cactina, Cactus and Cactus Grandiflorus and to our friendly old fraud Tongaline and its varied family of substitute frauds.

Write for a copy of this little work if you have not seen it and have not heretofore been interested in that branch of medicine and have your eyes opened to many of the little rascally tricks going on around you in the interests of the patent medicine fakirs.

Medical and Surgical Reports of the Protestant Episcopal Church in Philadelphia, Volume 1 edited by Astley P. C. Ashhurst, M. D. Cloth 406 pages finely illustrated.

This volume is rich in the statistical matter of large hospital experience and work and in addition contains many special articles by the hospital staff among which may be noted one on fractures of the forearm, the radius and allied fractures and dislocations. An article of great detail dealing with the Rational Treatment of Tetanus which is most exhaustive and thorough.

The original matter in this book is good but it will probably be considered that it would have been better for the profession had it been entitled The Clinics of The Episcopal Hospital in Philadelphia with an elimination of all statistical matter as to number of cases treated, etc. This would, of course, no longer make it a report in the true sense, but it would have eliminated considerable matter for which the busy physician does not care.

C. V. Mosby Company, St. Louis, have recently issued a very complete catalogue of the medical works issued from their press and other medical publications for which they are agents.

The edition is handsomely gotten up containing a very interesting color plate from the new second edition of Tuley's book on the "Diseases of Children." The catalogue will be sent free of charge to those physicians requesting it.

BIOLOGICAL PRODUCTS AND HOW TO USE THEM.

Journal of Oklahoma State Medical Association,
Muskogee, Oklahoma.

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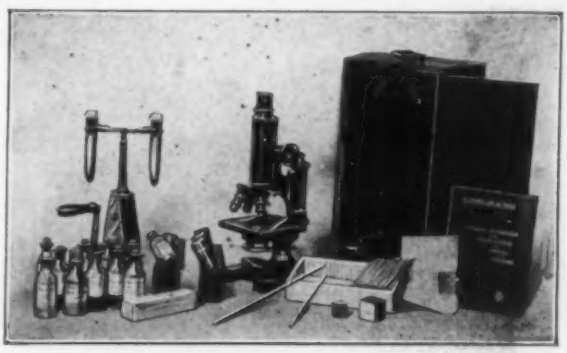

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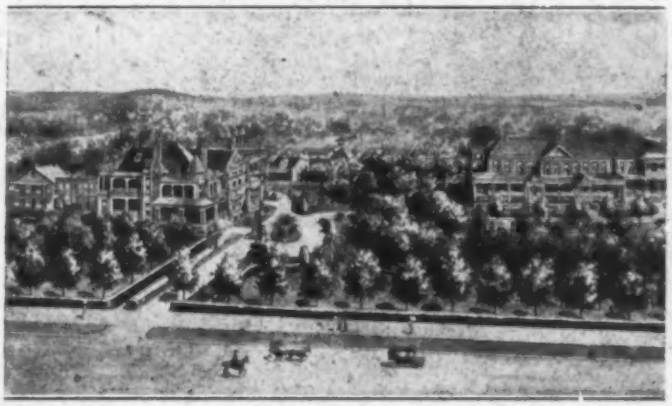

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References: The Medical Profession of Kansas City.

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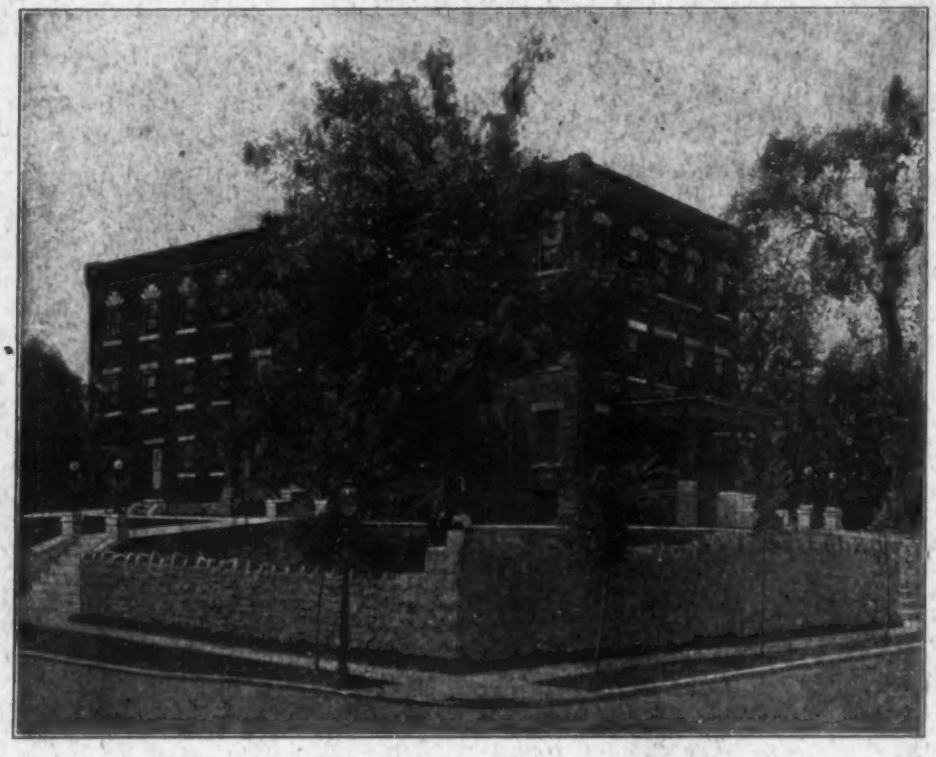

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